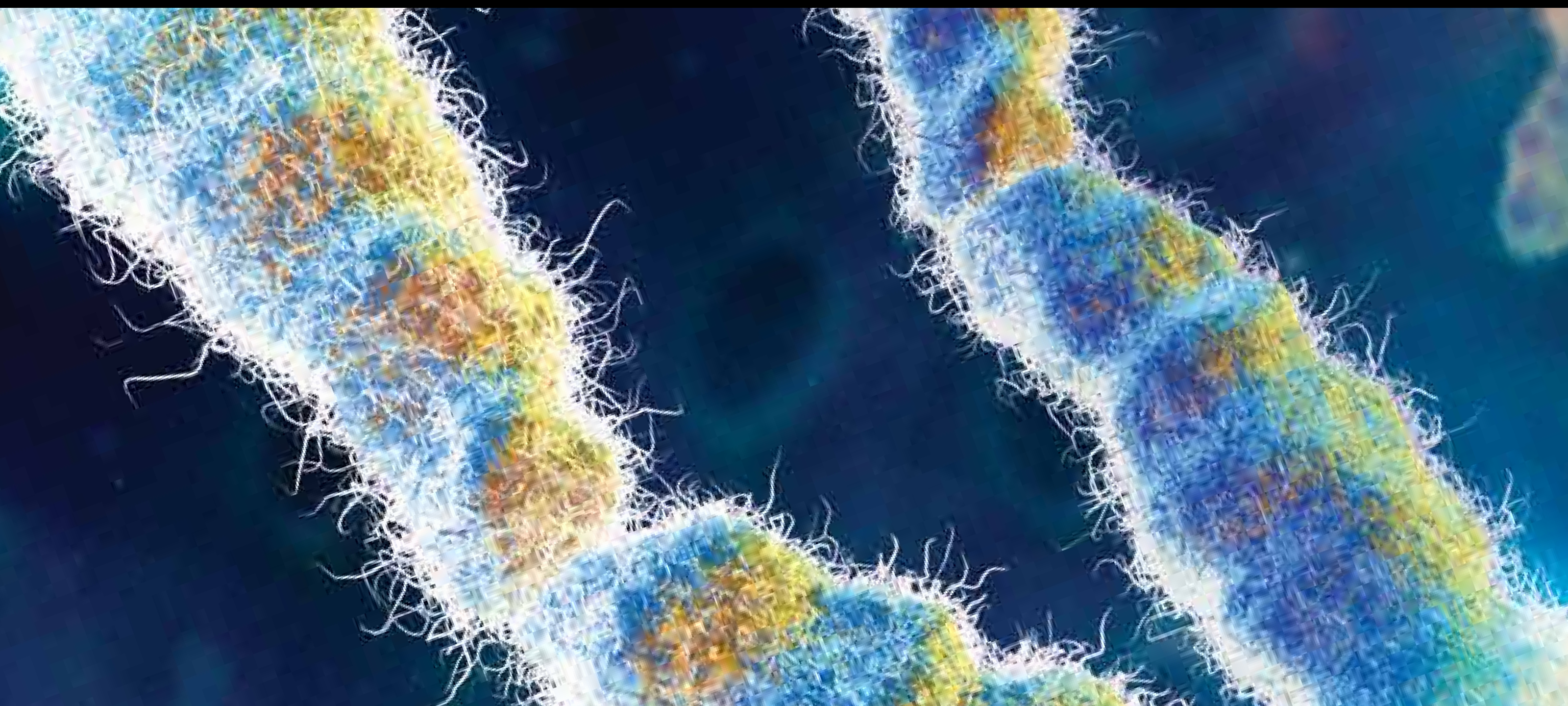
Journal of Aging Research

Aging, Physical Activity, and Disease Prevention 2012

Guest Editors: Iris Reuter, Lynn Rochester, Barbara Tettenborn, and Alice Nieuwboer



Aging, Physical Activity, and Disease Prevention 2012

Journal of Aging Research

Aging, Physical Activity, and Disease Prevention 2012

Guest Editors: Iris Reuter, Lynn Rochester, Barbara Tettenborn, and Alice Nieuwboer

Editorial Board

Contents

Hindawi Publishing Corporation
Journal of Aging Research
Volume 2012, Article ID 373294, 4 pages
doi:10.1155/2012/373294

Editorial

Aging, Physical Activity, and Disease Prevention 2012

Iris Reuter

Department of Neurology, Justus-Liebig University Giessen, 35392 Giessen, Germany

Correspondence should be addressed to Iris Reuter, iris.reuter@neuro.med.uni-giessen.de

Received 9 November 2012; Accepted 9 November 2012

Sociodemographic changes have led to an increase of aging in the society [1]. The biological and physiological changes of aging are primarily associated with a decline in muscle mass ranging from 1% to 2% per year past the age of 50, strength, endurance, and the inability to maintain balance [2]. Furthermore, the prevalence and incidence of cardiovascular diseases, diabetes mellitus, osteoarthritis, and neurodegenerative diseases rise with age resulting in a slowing of movements, imbalance, immobility, falls, and disability. Recent studies revealed an increase of disability among the older population [3, 4]. Age older than 84 years, lower education levels, obesity, comorbid conditions, not practicing physical activity, and sleeping more than 8 hours per day have been associated with higher disability [4]. Some studies have shown that elderly women are less active as well. Self-perceived health is worse in subjects with a greater number of comorbid conditions and disability and is considered a barrier for participation in exercise programmes [5]. Age of 80 years and beyond, more than 2 comorbid chronic conditions, and obesity have been shown to be associated with a lower likelihood of practicing leisure time physical activity [6]. Similarly, older people (age ≥80 years), those taking a greater number of medications for chronic conditions, obese, and with worse self-perceived health status tended to have a relatively lower physical fitness [5].

Besides health conditions, other factors might also affect physical activity. Some studies have shown that people who were physically active throughout their life keep the habit of exercising in old age. However, the time potentially available for leisure time physical activity depends on the amount of time required for paid employment, family, and daily mobility requirements. The time budget seems to be the most limited for middle-aged adults with job and family. Thus, many middle-aged adults give up leisure time sports and do not start participating in sports again [7, 8]. A sedentary life style is an independent risk factor for cardiovascular diseases, diabetes mellitus and musculoscelettal disorders [9].

There is a growing body of literature showing that regular exercise benefits well-being and health condition and leads to an increasing quality of life. Physical activity (PA) has been considered one key element for determining health status [3, 10]. Furthermore, physical activity improves mood and cognitive function [11]. Older adults who are physically active are c. 21% ($P \leq .05$) less likely than their counterparts to be diagnosed with dementia. Thus, vigorous physical activity may reduce the risk for dementia independently of other risk factors. The preventive character of physical activity for neurodegenerative disorders such as Parkinson's disease is not clear yet. While Chen et al. (2005) [12] and Thacker et al. (2008) [13] reported a lower risk to fall sick with Parkinson's disease, Logroscino et al. (2006) [14] did not find an effect of exercise on the risk of PD. However, there is growing evidence that patients suffering from PD benefit from exercise therapy [15].

An active life style also influences poor habits such as smoking, alcohol consumption, and fat nutrition.

Physical activity is defined as any bodily movement produced by skeletal muscles that result in energy expenditure and encompasses both leisure time activity (sports, exercise) [16] and activities of daily life [17]. The WHO has published guidelines for elderly in order to improve cardiorespiratory and muscular fitness [18], bone and functional health, depression, and cognitive decline. According to the WHO, physical activity includes leisure time physical activity, transportation (e.g., walking or cycling), occupational (if the individual is still engaged in work), household chores, play, games, sports, or planned exercise, in the context of daily, family, and community activities. Older adults should be engaged at least in 150 minutes aerobic physical activity of moderate intensity or 75 min of vigorous physical activity throughout the week. To obtain additional health benefits, older adults should increase their moderate intensity of aerobic physical activity to 300 minutes per week, or engage in 150 minutes of vigorous intensity aerobic physical activity per week, or an equivalent combination of moderate and vigorous intensity activity. It has been shown that 20–30 min moderate intensity physical activity on most days result in better physical functions in older adults [19].

The minority of healthy elderly people meet these criteria for activity and even fewer older adults with concomitant diseases exercise regularly with sufficient intensity and frequency [20].

Although there are studies showing an increase in leisure time physical activity among elderly in Spain [5], the increase of physical activity did not lead to an increased physical fitness.

It is still an ongoing debate whether a certain intensity of exercises is necessary to obtain a morbidity lowering effect for cardiovascular and cerebrovascular accidents. While some researchers claim that low intensity exercise such as regular walking, biking, or gardening have a preventive effect [21], other study results indicate that the preventive effect of exercising depends on the intensity of the exercise [22] and an increase of physical fitness.

According to Lee the additional use of on average 3000–35000 kcal lowers mortality significantly [23]. Some authors found a graded dose response of the volume of physical activity with all-cause mortality, stroke, and several coronary heart disease factors [24]. A clear dose-response between the exercise volume as measured by MET-min × week (−1) and VO_2max and between the intensity of physical activity and the VO_2max response has been shown.

However, the energy expenditure of leisure time physical activity did not correlate with the risk of decline in perceived health [21].

Exercise therapy has now been widely accepted as useful tool in the prevention and treatment of several diseases, thus this special issue on aging, physical activity, and disease prevention will now appear annually. Comparable to the first special issue on aging, physical activity and age prevention the current issue covers a wide range of topics. Ten papers have been accepted for publication.

One paper investigates the role of exercise therapy in the prevention of decline in aging muscle function and in the prevention of glucocorticoid myopathy and muscle unloading. The paper reviews the effects of muscle wasting and the possibilities of exercise therapy. The authors explore the potential molecular effects of exercise therapy on muscle protein metabolism. They postulate that strength exercise can change the renewal of contractile proteins in accordance with the needs of muscle contractile apparatus and endurance exercise might restore the oxidative capacity by stimulation of mitochondrial biogenesis.

Thus, both, strength and endurance exercise seem to be promising tools for aging-related disease prevention.

Another paper assesses the correlation between physical activity and frailty phenotypes in females with Parkinson's disease. The authors found that nonfrail patients recorded more physical activity than frail, self-reported physical activity was greater in PD patients than in non-PD subjects. However, physical activity was related to frailty in non-PD subjects only. In PD patients, frailty was rather related to disease associated factors; hence, disease management might be more important.

The impact of positive and negative social control, support, and perceived strain on physical activity is discussed in another paper. Hierarchical regression analyses revealed that perceived support and perceived strain were not correlated with physical activity. However, age and sex interacted with social control, such that more positive social control was associated with more frequent physical activity for younger men, while more positive and negative social control were significantly associated with less frequent physical activity for older men. There was no association between social control and physical activity among women. The authors concluded that health professionals and social partners should be discouraged from using negative social control because these strategies may be ineffective for women and younger men and may be counter productive for older men. Positive social control strategies might be appropriate for young men and alternative strategies have to be pursued for women.

The authors of another paper provide an overview of "*Convivência*" groups in Brasil. The paper reports on the results of a survey conducted in Florianópolis. Social groups can be crucial for health and well-being in old age. A variety of programmes for older adults supported by the Brazilian National Public Policy in 2003 and established since 2002 were designed to enhance social activities among the older adult population. Community-based social groups known as "convivência" groups were extremely popular. Participating in "convivência" groups helped elderly to be socially engaged and to live actively.

The purpose of one of the papers was to quantify the extent to which physical activity differed between Veterans and non-Veterans and to determine how diabetes and age influenced this association. After adjusting for age, sex, race and ethnicity, household income, education level, body mass index (BMI), and recent health checkup, Veteran status was associated with a small but significantly larger amount of average weekly moderate physical activity. Diabetes and prediabetes were associated with significantly lower mean levels of both moderate and vigorous intensity physical activity as was increasing age. Veteran status had no impact on the association between diabetes, age, and physical activity.

There is a paper that provides a review on the increase of life expectancy by physical exercise. The authors performed a systematic PubMed search on life expectancy in physically active and inactive subjects, in addition articles comparing life expectancy of athletes compared to that of nonathletes were reviewed. Physical activity reduces many major mortality risk factors including arterial hypertension, diabetes mellitus type 2, dyslipidemia, coronary heart disease, stroke, and cancer. All-cause mortality is decreased by about 30 to 35% in physically active as compared to inactive subjects. The studies suggest that regular physical activity is associated with an increase of life expectancy by 0.4 to 6.9 years. Aerobic endurance athletes showed a greater life expectancy, but it remains unclear if high-intensity sports activities further increase life expectancy.

The meaning of aging and the development of osteoarthritis is highlighted in another paper. Osteoarthritis (OA) is a major health burden leading to progressive pain and reduced mobility with age as the most prominent risk factor for the development and progression of OA. Inflammatory cytokines play a role in the development of osteoarthritis. Joint movement has been shown to exhibit anti-inflammatory mechanisms. Therefore, physical activity or physiotherapy in the elderly might reduce inflammatory processes and increase muscle mass.

The surgical treatment of end-stage osteoarthritis in elderly patients was the focus of one of the papers. The authors emphasized that elderly patients may benefit more by total ankle replacement (TAR) than by the alternative ankle arthrodesis, since rehabilitation after TAR is easier than that after ankle arthrodesis. Immobilisation and protection time is shorter and articular and muscle function less affected. Total ankle replacement might be an option to regain mobility and quality of life in elderly patients.

There is a paper that explores the role of barriers as limiting factor to participation in physical activity in Canadian seniors. The identification of barriers to physical activity and exercise has been used for many decades to explain exercise behaviour in older adults. Typically health concerns are the number one barrier to participation. In contrast to earlier results the current research did not identify a health condition limitation, illness, or injury as a barrier to participation in physical activity. Barriers are not the limiting factor and physical activity programming has to be focused on the health needs of our aging population.

Another paper reports on the efficacy of a multimodal cognitive rehabilitation programme including psychomotor and endurance training in Parkinson's disease. Executive dysfunction and dementia are mayor problems in Parkinson's disease and more disabling than motor disturbances. The aim of the study was to compare three different cognitive training programmes. The results showed that the multimodal cognitive rehabilitation programme which included physical exercises has been more successful than the other cognitive training programmes. In addition some translation of the improvements into real life has been obtained.

Iris Reuter

References

[1] United Nations, "Population Division: World Population Ageing 1950–2050," New York, NY, USA, 2002.

[2] K. Sakuma and A. Yamaguchi, "Sarcopenia and age-related endocrine function," *International Journal of Endocrinology*, vol. 2012, Article ID 127362, 10 pages, 2012.

[3] T. E. Seeman, S. S. Merkin, E. M. Crimmins, and A. S. Karlamangla, "Disability trends among older Americans: National Health and Nutrition Examination surveys, 1988–1994 and 1999–2004," *American Journal of Public Health*, vol. 100, no. 1, pp. 100–107, 2010.

[4] D. Palacios-Ceña, R. Jiménez-García, V. Hernández-Barrera, C. Alonso-Blanco, P. Carrasco-Garrido, and C. Fernández-de-Las-Peñas, "Has the prevalence of disability increased over the past decade (2000–2007) in elderly people? A Spanish population-based survey," *Journal of the American Medical Directors Association*, vol. 13, no. 2, pp. 136–142, 2012.

[5] D. Palacios-Ceña, C. Fernandez-de-Las-Peñas, V. Hernández-Barrera, R. Jiménez-Garcia, C. Alonso-Blanco, and P. Carrasco-Garrido, "Sports participation increased inSpain: a population-based time trend study of 21 381 adults in the years 2000, 2005 and 2010," *British Journal of Sports medicine*, vol. 46, no. 16, pp. 1137–1139, 2012.

[6] T. S. Church, C. P. Earnest, J. S. Skinner, and S. N. Blair, "Effects of different doses of physical activity oncardiorespiratory fitness among sedentary, overweight or obese postmenopausal women with elevated blood pressure: a randomized controlled trial," *Journal of the American Medical Association*, vol. 297, no. 19, pp. 2081–2091, 2007.

[7] World Health Organisation, *Health and Development through Physical Activity and Sport*, World Health Organization, Geneva, Switzerland, 2003.

[8] K. N. Boutelle, D. M. Murray, R. W. Jeffery, D. J. Hennrikus, and H. A. Lando, "Associations between exercise and health behaviors in a community sample of working adults," *Preventive Medicine*, vol. 30, no. 3, pp. 217–224, 2000.

[9] H. Löllgen, "Also true in coronary heart disease: the well trained patient lives longer," *MMW Fortschritte der Medizin*, vol. 144, no. 49, p. 5, 2002.

[10] T. Prohaska, E. Belansky, B. Belza et al., "Physical activity, public health, and aging: critical issues and research priorities," *Journals of Gerontology B*, vol. 61, no. 5, pp. S267–S273, 2006.

[11] S. Colcombe and A. F. Kramer, "Fitness effects on the cognitive function of older adults: a meta-analytic study," *Psychological Science*, vol. 14, no. 2, pp. 125–130, 2003.

[12] H. Chen, S. M. Zhang, M. A. Schwarzschild, M. A. Hernán, and A. Ascherio, "Physical activity and the risk of Parkinson disease," *Neurology*, vol. 64, no. 4, pp. 664–669, 2005.

[13] E. L. Thacker, H. Chen, A. V. Patel et al., "Recreational physical activity and risk of Parkinson's disease," *Movement Disorders*, vol. 23, no. 1, pp. 69–74, 2008.

[14] G. Logroscino, H. D. Sesso, R. S. Paffenbarger Jr., and I. M. Lee, "Physical activity and risk of Parkinson's disease: a prospective cohort study," *Journal of Neurology, Neurosurgery and Psychiatry*, vol. 77, no. 12, pp. 1318–1322, 2006.

[15] I. Reuter and G. Ebersbach, "Effektivität von Sport bei M. Parkinson," *Aktuelle Neurologie*, vol. 5, pp. 236–247, 2012.

[16] C. J. Caspersen, K. E. Powell, and G. M. Christenson, "Physical activity, exercise and physical fitness: definitions and distinctions for health-related research," *Public Health Reports*, vol. 100, no. 2, pp. 126–131, 1985.

[17] M. C. Ashe, W. C. Miller, J. J. Eng, L. Noreau, and Physical Activity and Chronic Conditions Research Team, "Older adults, chronic disease and leisure-time physical activity," *Gerontology*, vol. 55, no. 1, pp. 64–72, 2009.

[18] "Global recommendation on physical activity for health 65 years and above," World Health Organisation, 2011.

[19] J. S. Brach, E. M. Simonsick, S. Kritchevsky, K. Yaffe, and A. B. Newman, "The association between physical function and lifestyle activity and exercise in the health, aging and body composition study," *Journal of the American Geriatrics Society*, vol. 52, no. 4, pp. 502–509, 2004.

[20] S. C. Dumith, P. C. Hallal, R. S. Reis, and H. W. Kohl III, "Worldwide prevalence of physical inactivity and its association with human development index in 76 countries," *Preventive Medicine*, vol. 53, no. 1-2, pp. 24–28, 2011.

[21] L. DiPietro, "Physical activity in aging: changes in patterns and their relationship to health and function," *Journals of Gerontology A*, vol. 56, no. 2, pp. 13–22, 2001.

[22] J. E. Manson, F. B. Hu, J. W. Rich-Edwards et al., "A prospective study of walking as compared with vigorous exercise in the prevention of coronary heart disease in women," *The New England Journal of Medicine*, vol. 341, no. 9, pp. 650–658, 1999.

[23] I. M. Lee, C. C. Hsieh, and R. S. Paffenbarger, "Exercise intensity and longevity in men: The Harvard Alumni Health Study," *Journal of the American Medical Association*, vol. 273, no. 15, pp. 1179–1184, 1995.

[24] P. Oja, "Dose response between total volume of physical activity and health and fitness," *Medicine and Science in Sports and Exercise*, vol. 33, no. 6, supplement, pp. S428–S437, 2001.

Hindawi Publishing Corporation
Journal of Aging Research
Volume 2012, Article ID 235765, 15 pages
doi:10.1155/2012/235765

Clinical Study

Efficacy of a Multimodal Cognitive Rehabilitation Including Psychomotor and Endurance Training in Parkinson's Disease

I. Reuter,[1] S. Mehnert,[1] G. Sammer,[2] M. Oechsner,[3] and M. Engelhardt[4]

[1] *Department of Neurology, Justus-Liebig University, Klinikstraße 33, 35392 Giessen, Germany*
[2] *Department of Psychiatry, Cognitive Laboratory Justus-Liebig University, Am Steg 22, 35385 Giessen, Germany*
[3] *Neurologisches Rehabilitationszentrum, HELIOS Klinik Zihlschlacht AG, Hauptstrße 2-4, 8588 Zihlschlacht, Switzerland*
[4] *Department of Orthopedic Surgery, Klinikum Osnabrück, Am Finkenhügel 1, 49076 Osnabrück, Germany*

Correspondence should be addressed to I. Reuter, iris.reuter@neuro.med.uni-giessen.de

Received 1 April 2012; Revised 19 June 2012; Accepted 3 July 2012

Academic Editor: Alice Nieuwboer

Mild cognitive impairment, especially executive dysfunction might occur early in the course of Parkinson's disease. Cognitive training is thought to improve cognitive performance. However, transfer of improvements achieved in paper and pencil tests into daily life has been difficult. The aim of the current study was to investigate whether a multimodal cognitive rehabilitation programme including physical exercises might be more successful than cognitive training programmes without motor training. 240 PD-patients were included in the study and randomly allocated to three treatment arms, group A cognitive training, group B cognitive training and transfer training and group C cognitive training, transfer training and psychomotor and endurance training. The primary outcome measure was the ADAS-Cog. The secondary outcome measure was the SCOPA-Cog. Training was conducted for 4 weeks on a rehabilitation unit, followed by 6 months training at home. Caregivers received an education programme. The combination of cognitive training using paper and pencil and the computer, transfer training and physical training seems to have the greatest effect on cognitive function. Thus, patients of group C showed the greatest improvement on the ADAS-Cog and SCOPA-COG and were more likely to continue with the training programme after the study.

1. Introduction

Idiopathic Parkinson's disease (PD) is a neurodegenerative disorder characterized by loss of dopaminergic neurons and basal ganglia dysfunction. The prevalence of PD increases with age and is estimated in 100–200/100000 people [1, 2] worldwide. The clinical hallmarks of PD are akinesia, rigidity, and tremor [3, 4]. However, a spectrum of nonmotor symptoms occurs in Parkinson's disease. One of the most disabling symptoms is dementia, which is common among patients with PD with an average prevalence of 40% in cross-sectional studies and a cumulative prevalence approaching 80% [5, 6]. PD dementia is the third most common reason for dementia and is associated with rapid functional and motor decline, shortened survival [7], greater sensitivity to medication, higher risk of developing psychosis, reduced quality of life for both patients [8] and caregivers [9], increased caregivers' stress, and frequent transfer to nursing homes [10]. Clinical characteristics of PD dementia are cognitive slowing, executive deficits, visuospatial deficits, and memory impairments [11]. Pathological findings include Lewy bodies outside the substantia nigra, neurofibrillary tangles, and amyloid plaques [12]. Neurochemically, cholinergic deficits are found to be the most consistent associated with cognitive and neuropsychiatric symptoms [13].

In contrast to dementia, mild cognitive impairment (MCI) might occur early in the course of PD. Approximately, a quarter of PD-patients without dementia have mild cognitive impairment (PD-MCI). The Movement Disorder Society commissioned task force reported that MCI in PD is associated with increasing age, increasing disease duration, and disease severity [14]. However, 20% of PD-patients might have MCI at the time of diagnosis [15]. The clinical profile of PD-MCI is heterogeneous with a range of cognitive domains affected. Nonamnestic, single-domain impairment is the most common subtype of PD-MCI [14]. Criteria for the diagnosis of PD-MCI have been published recently [14]. Neuropsychological testing should include two tests within each of the five cognitive domains (attention, working memory, executive language, memory, and visuospatial). A diagnosis of PD-MCI impairment has to be found either in two neuropsychological tests in one domain or one impaired test in two different domains. Components of the executive systems are attention (focusing on relevant information), selective visual attention, inhibition (inhibition of irrelevant information) [15], overcoming of strong habitual responses or resisting temptation [16], task and time management, monitoring and coding of information for processing in the working memory, flexibility, set maintenance, and set shifting. The executive system can be viewed as a manager enabling the adaptation of the perceptive, cognitive, and motor system to new tasks [17]. Thus, patients with impaired executive functions face many difficulties in everyday life. They have a low attention span, difficulties in problem solving and decision making, in dual tasking, in set shifting, in visuospatial tasks, in adaptation to new tasks, and even in verbal learning and delayed recall. For example, PD-patients with impairment of executive functions may have difficulties in simultaneously driving a car and searching for a street or in keeping appointments. Executive dysfunctions also affect social components and the interaction with other people [15]. Patients are reported of being more irritable and having difficulties in suppressing inappropriate behaviour. PD-patients with MCI might have a higher risk to develop PD dementia. There is some evidence from previous studies that the presence of a nonamnestic single-domain MCI- subtype, executive deficits, impaired verbal fluency, visuospatial deficits, memory and language dysfunction predict PD-dementia, since patients with mild cognitive impairment have a higher risk of developing dementia. Intervention at an early stage of cognitive decline and prevention of the progress from MCI to dementia would be desirable. While treatment of motor symptoms has largely improved, treatment of cognitive dysfunction is still limited. Acetylcholine esterase inhibitors improve cognitive functioning only in some patients. Furthermore, transfer of improvements in cognitive training into activities of daily living has been extremely difficult. There is a large body of studies on animals and humans in the literature showing positive effects of exercise and sports on cognition [18–23]. Several studies suggest an enhancement of cortical plasticity by exercise [24, 25]. Executive functioning [19, 20, 26] as well as quality of daily living [27, 28] was found to be improved by aerobic endurance exercise. Patients, who complain of cognitive problems, suffer more often from cognitive deficits than patients without complaints [29]. Therefore, these patients should be offered neuropsychological testing. We have chosen a comprehensive training approach and designed a study using a multimodal cognitive training to improve cognitive functions.

The aim of the present study was to compare the effect of a multimodal cognitive training regime including paper and pencil tasks combined with transfer tasks and a psychomotor training with a cognitive training based on paper and pencil tasks only and a cognitive training consisting of various tasks requiring executive functions combined with transfer tasks.

2. Methods

2.1. Subjects. 240 patients, men and women who were between 50 and 80 years old and who had received a diagnosis of Parkinson's disease according to the UK brain bank criteria [4], were recruited for the study at the Hospital for Parkinson's Disease Bad Nauheim. Patients had been admitted at the hospital for rehabilitation. The presence of MCI was required for inclusion into the study. MCI was diagnosed when (a) patients complained of cognitive decline, preferably corroborated by a reliable source, (b) minimal effect on day-to-day functioning and the absence of dementia, and (c) presence of cognitive abnormalities which cannot be simply attributed to age. Exclusion criteria were severe concomitant diseases, which limit physical performances, a second neurodegenerative disease, and lack of motivation to improve cognitive deficits and the presence of dementia. De novo patients and those who had undergone surgery for DBS were not included into the study. All patients were assessed by a movement disorder specialist. Medical treatment was optimised prior to the study. It was aimed at keeping medication stable during the study.

2.2. Design. The study was divided into two periods. Patients were enrolled into the study during an in-patient stay on a rehabilitation ward. The first part of the study (4 weeks) took place on the rehabilitation unit with a supervised cognitive training conducted by physiotherapists, occupational therapists, and two neuropsychologists.

Patients were randomly allocated to one of the three training groups. Randomisation was conducted by using a computer-generated sequence. All groups received a cognitive training regime using paper and pencil material and a multimedial PC training. Group A received cognitive training only, while group B took part in a transfer training and a cognitive training. Group C conducted a cognitive training, and transfer and psychomotor trainings. Patients of group A and B had additional relaxation training and occupational training without transfer training to compensate for the additional training time of group C.

After randomisation medical history was taken, patients underwent medical and neurological assessments and were asked about their cognitive difficulties. They identified cognitive deficits that they want to improve. Severity of Parkinson's disease was assessed by using the Unified Parkinson's Disease Rating Scale (UPDRS) [30].

Demographic data included information about age, body mass index (BMI), duration of disease, weekly sports activity, smoking habits, medication and concomitant diseases (hypertension, chronic obstructive pulmonary disease, thyroid disease, diabetes mellitus, hypercholesterinaemia, and osteoarthritis), education, profession, family, onset and severity of disease, history of psychosis, and impairments in daily living. Patients kept an activity log one week prior to the training programme and one week prior to the third assessment. Sports activities, time spent sitting, and doing light, moderate, and heavy work were recorded. The ethical committee of the Justus-Liebig University has approved the study and all patients have given written informed consent. Figure 1 shows the study design.

2.3. Scales Used for Neurological and Neuropsychological Assessment of PD. For the assessment of the longitudinal course of the disease, the Unified Parkinson‘s disease rating scale (UPDRS) was applied [30].

For the assessment of the goals of the cognitive training, the Goal Attainment Scale was chosen. The Goal Attainment Scaling (GAS) was used to define individual realistic and feasible goals according to patients' needs and expectations. The cognitive difficulties of the patients were assessed and the results of the baseline test were explained to the patients. In this study, GAS was measured using a 6-point Likert scale −3 represented function that is worse than at the start of treatment, −2 no change, −1 some improvement without meeting the expected goal, 0 represented goal achievement and +1 or +2 overachievement (exceeding the defined therapeutic goal) [31].

2.3.1. Neuropsychological Tests. At the beginning of the study, all patients underwent two cognitive screening tests (PANDA and MMSE) [32, 33], followed by a detailed cognitive test battery including the ADAS-COG subscale and the SCOPA-COG as primary and secondary outcome measures, respectively. The neuropsychological assessment was repeated after the 4-week inpatient stay and after the 6-month training at home.

2.3.2. ADAS-COG (Alzheimer Assessment Scale Cognition). Although the ADAS-COG is the primary outcome measure in many clinical trials [34, 35], it is not a specific test for cognitive impairment in Parkinson's Disease. The scale was chosen as primary outcome measure in the current study, because it was the primary outcome measure in earlier trials assessing effects of medication on cognitive function in PD [6]. The study by Emre et al. [6] gave an estimate regarding the relevance of a 3-point improvement on the ADAS-COG in PD. Thus, it was possible to relate the results of the current study to previous results.

The conceptual framework underlying the ADAS-COG is to identify three reproducible factors: memory, language, and praxis [35]. The ADAS-COG score ranges in total from 0 to 70 points with higher scores indicating greater impairment.

2.3.3. SCOPA-COG (Scales for Outcome of Parkinson's Disease-Cognition). The SCOPA-COG is an instrument which was designed to assess the specific cognitive deficits found in Parkinson's disease [36]. The scale consisting of 10 items covers the following domains: memory and recall (verbal recall, digit span backward, and indicate cubes), attention (counting backward, months backward), executive function (fist-edge-palm, semantic fluency, and dice), visual-spatial functions (assembly pattern), and memory (delayed recall). The score ranges from 0 to 43 points with higher scores reflecting better performance.

2.3.4. Additional Tests. Since PD-patients show pronounced deficits in executive functions additional tests for evaluation of executive functions were conducted at baseline, second and final assessment: Information processing speed (Paced auditory serial addition test (PASAT) [37]), Executive function (Behavioural assessment of the dysexecutive syndrome (BADS) [38]), Test for premorbid performance (Mehrfach-Wortschatz-Test (MWT-B) Multiple choice word test [39]), Assessment of mood and anxiety (Hospital anxiety and depression scale was applied [40]).

2.3.5. Health-Related Quality of Life (Parkinson's Disease Questionnaire 39 (PDQ-39)). For assessment of health-related quality of life, patients filled in the PDQ-39 [41]. It consists of 8 subscales. The sum score of raw data ranges from 0 to 156 points, with high scores indicating lower health-related quality of life. For better comparison of the results, raw data were transformed and expressed in percentages of maximal possible sum score.

The assessments were performed by psychologists and movement disorders specialists who were blinded to the treatment allocation of the patients.

2.4. Training Programmes. Patients performed the training programmes in onstage and after optimisation of the medication. Dopaminergic deficits may affect the performance of patients in cognitive and physical exercises [42, 43].

2.4.1. Cognitive Training. The cognitive training programme was individually tailored to patients' requirements based on the results of the baseline tests. Four individual (one-to-one) 60 min-lessons took place each week. All patients received at least 14 cognitive training sessions.

The training included training of attention, concentration, biographical work, reasoning, memory, working memory, social rules, anticipation, cognitive information speed, prospective memory, cognitive estimation, problem solving, sequencing and planning, associations, and coping with disease.

For the training programme, a set of tasks requiring executive and memory functions was chosen from a variety of specific tests. Executive tasks of the BADS, which were not used for the baseline assessment, were included in the training. Simple patterns of the "Raven's Progressive Matrices" were used to establish problem solving strategies in the patients. Picture arrangement tasks, picture completion tasks, block design, and object assembly were adapted from the "Wechsler Intelligence test for children." For improvement of verbal fluency, patients were encouraged to tell short stories or discuss short text passages. Photos were used for training of working memory. Tasks including visual search and rule finding were practised by using a PC-based programme. The training methods were designed to improve the various cognitive deficits, diagnosed at baseline, and focused on the executive functions. Task difficulty was adapted to the individual performance level of the patients. Table 1 shows the content of the cognitive training and the percentage of time spent with different tasks.

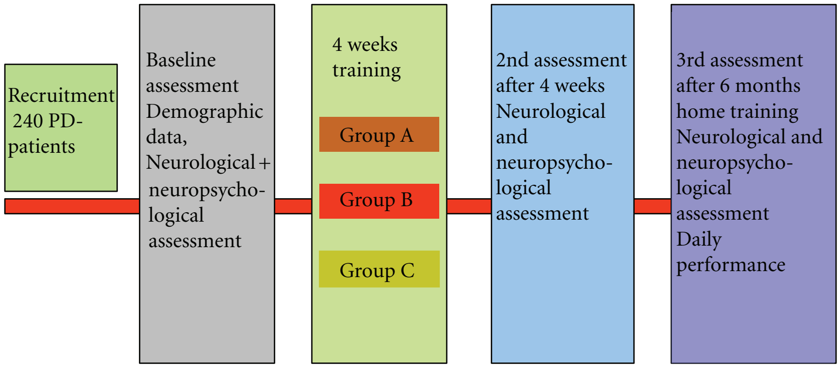


Figure 1: Study design group A: Cognitive training; group B: Cognitive + transfer training, group C: Cognitive, transfer + motor training.

2.4.2. Transfer Training. The aim of the training was to support patients to manage their daily life better and to become more self-confident. Therefore, patients were asked to practise competence in tasks of daily routines. The transfer training programme was composed according to the baseline test results. Special preferences of the patients were considered. The transfer training included training of concentration, use of mnemonics, strategy (planning), navigational skills, impulse control, decision processes, listening training and memory, behaviour, calculating, handling of money, summarising of articles read or heard, and decision making. Typical tasks were to find the way to the supermarket or to prepare a meal, to go to the bank, pay a bill, and to use mnemonics. Patients had to look after a vegetable patch and some flowers to improve prospective memory. For better evaluation of the training, tasks were allocated to different categories: concentration, strategy, orientation, planning, and the use of mnemonic devices. The training took place 3 times a week, each lasted 90 min. Patients received at least 10 sessions of transfer training (Table 2).

2.4.3. Motor Training. Group C performed a motor training resembling motor skill training or psychomotor training applied in children. The training included games and tasks designed to enhance inhibitory control, working memory, attention, visuospatial abilities, and planning and motor skills [44–47]. The training should also improve coordination, strength, speed, perception, and orientation. Patients should improve perception of their body. The therapeutic approach was based on individual capabilities and needs. Patients learned to perform motor sequences, dual tasking (walking and bouncing or throwing a ball), and spatial orientation tasks (finding items, remember hidden items). They walked through a parcours with obstacles following changing rules in order to improve anticipation and mental flexibility. For improvement of time estimation and movement performance mental imagery was used. Patients had to follow the guidance of the physiotherapist given by touch at different parts of the body. In addition, an aerobic training which should be of benefit for executive functions was performed. The training was conducted partly outdoors with inclusion of Nordic walking in summer. In winter patients walked on a treadmill. The training included at least 10, maximal 12 sessions each lasting 60 minutes.

2.5. Education of Caregivers. A long lasting training effect depends on continuing training. Thus, cognitive training and exercises need to be adapted to the home environment. Accordingly, the caregivers most often the patients' family were included into the programme. The education for the caregivers consisted of 5 modules: information about Parkinson's disease, psychological aspects and the role of a caregiver, information about help aids, information on care management, assessment of individual problems, support in cognitive (all groups) and transfer training (groups A and B), NW and movement skill training. Course instructors were a specialist nurse, a physiotherapist and a psychologist.

Phase II Continuation of Training at Home. Corresponding to the allocation to the training groups patients got written instructions for the cognitive training, transfer training, and physical exercises at home. The instructions encompassed a collection of the tasks conducted during the stay at the hospital. Caregivers were advised how to organise the training but the hospital staff did not organise the training at home. All patients were asked to perform three 45 min cognitive training sessions per week using paper, pencil, and a computer programme. Patients of groups B and C were asked to continue with two transfer trainings per week and patients of group C got instructions to conduct movement skill training and aerobic training lessons twice a week. To compensate for the additional physical training of group C, patients of group A and B were provided with prescriptions for relaxation training.

2.6. Evaluation of the Training. All patients were tested using a neuropsychological test battery at three time points: prior to the training, and prior to discharge to assess the short-term effect, and 6 months after the training to assess the long-term effect.

Caregivers were asked regarding their own well being and regarding the cognitive competence of the patients in activities of daily living. Patients and caregivers kept a diary

Table 1: Cognitive training.

Training section	Examples for tasks
Planning strategies	Shopping lists, key search of the BADS subscales, to get items out of a bottle, special wooden 3-dimensional jig saw
Mnemonics	Using memory hooks, writing of suitable mnemonics, and planning where to place them
Decision making	Computer tasks, painting
Set shifting	To solve various tasks according to changing rules, to calculate for 5 min, then to read for 10 min and to write for 5 min, or to switch tasks after every third answer, categorising items according to different rules
Calculation	Calculation tasks
Navigational skills	Finding the way through a maze on a screen, finding the way around obstacles on a paper or on a screen
Information speed processing	Connecting numbers or letters, connecting figures to an image, and finding the meaning of a fictive word
Summary	Extracting the relevant information from news or from a story or a short movie
Concentration	Finding similar or dissimilar items

Table 2: Transfer training.

Tasks	Examples
Planning and sequencing	Preparing of meals, shopping, construction of items, repairing of items, and art works
Reasoning	Finding the right solution, using deductive strategies, finding a way by incomplete instructions, to find out how things work, and use of unfamiliar tools
Concentration	Sorting and selection tasks, to fit small items, to build a model of a castle, knitting and weaving patterns, jig saw, to listen to a story and to press a button, when a previously identified word was read
Memory	Role play, music performance, singing, and learning poems
Working memory	Games, n-back tasks, dual tasks like driving on a driving simulator and listening to the news, calculating during walking, to follow complex instructions, and to extract the necessary information out of a long text
Anticipation	Walking on a crowded sidewalk, watching a movie and predict what people will do next, finish a picture story, and decipher the mood of people on pictures
Pospective memory	To look after a vegetable patch and flowers, to keep appointments, and to take over special tasks at the beginning of the week
Cognitive information speed	Follow verbal instructions given with increasing content of information and speed, card games, and complex reaction tasks
Social rules	Define the appropriate behaviour in different situations, role play,
Association	Solving tasks by association, finding common features, and categorisation
Cognitive estimation	Estimation of height, quantity of items shown, and weight

to record training lesions. The diaries were collected and analysed at the 3rd assessment.

2.7. Statistical Analysis. Statistical analysis was conducted using IBM SPSS Statistics 18.0 (IBM, Somers, USA) statistical software. Formal power analysis was performed prior to the study. The power analysis was based on an improvement of the ADAS-COG by 3 points. The results indicated that a sample size of 60 subjects per group was sufficient. Since comprehensive training programmes including several assessments imply dropouts, a drop-out rate of 20% was taken into account. Demographic data on ordinal level were analysed by using a nonparametric test (Kruskall-Wallis). The Kruskall-Wallis test was also applied for the analysis of depression and the BADS subscales. Demographic continuous data were analysed by using One-way ANOVA. Linear model for repeated measures was used for analysis of training outcomes. The repeated measure analysis provides information about "between and within subjects" effects. Within subject effects give information about training effects over the assessment period. Linear trends showed if there was a systematic change of training effects over time. The interaction between groups and the linear trend of days (assessments) provided information about the difference in the rate of improvement between groups. The between subject factor compared the overall treatment effect between the groups. Post hoc analysis was done using Bonferroni tests. Parametric data were tested for normal distribution by using the Kolmogorov-Smirnov test. Significance level was set at 0.05.

3. Results

General Results, Demographic Data and Accomplishment of the Training. In total 223 patients (97.1%) completed the programme during the in-patient stay in the hospital: 71 (90%) patients in group A, 75 (93.8%) in group B, and 76 (95%) patients in group C. The patients were on average 64 ± 4 years old and that 8 years diagnosed with PD. The patients did not differ significantly in demographic data (Table 3).

Table 3: Demographic data.

	Group A $N = 71$			Group B $N = 75$			Group C $N = 76$		
Gender	F = 35		M = 36	F = 36		M = 39	F = 36		M = 40
Duration of PD (months) ∅± SD		98 ± 8			95 ± 9			100 ± 6	
Stage (Hoehn and Yahr)									
II		$N = 7$			$N = 6$			$N = 10$	
III		$N = 55$			$N = 59$			$N = 58$	
IV		$N = 9$			$N = 10$			$N = 8$	
Medication									
L-Dopa		Yes: $N = 68$			Yes: $N = 64$			Yes: $N = 59$	
Dopamine agonist		Yes: $N = 53$			Yes: $N = 56$			Yes: $N = 59$	
MA0 inhibitor		$N = 43$			$N = 38$			$N = 43$	
COMT inhibitor		$N = 33$			$N = 31$			$N = 34$	
Antidepressants		$N = 7$			$N = 8$			$N = 8$	
Neuroleptic drugs		$N = 5$			$N = 8$			$N = 7$	
Formal education (years) ∅± SD		10 ± 1.2			11 ± 0.6			11 ± 1.0	
Marital status m = married, s = single, and p = partner	m = 58	s = 9	p = 5	m = 61	s = 11	p = 3	m = 63	s = 9	p = 4
Home (own home, renting)	Own: $N = 40$		Renting: $N = 32$	Own: $N = 43$		Renting; $N = 32$	Own: $N = 40$		Renting: $N = 36$
BMI ∅± SD		27.5 ± 4			26.8 ± 7			27.2 ± 3	
Smoking	Yes: $N = 7$		No: $N = 65$	Yes: $N = 10$		No: $N = 65$	Yes: $N = 9$		No: $N = 67$
Sports activities (min)/week ∅± SD		155 ± 17			163 ± 25			147 ± 17	
Physical work h/week ∅± SD									
Very hard		8.5 ± 2.6			9.2 ± 2.8			9.8 ± 2.1	
Hard		15.5 ± 4.5			14.9 ± 5			15.1 ± 5.5	
Comorbidity									
Coronary heart disease		$N = 7$			$N = 6$			$N = 8$	
Hypertension		$N = 32$			$N = 33$			$N = 36$	
Diabetes mellitus		$N = 7$			$N = 10$			$N = 8$	
COPD		$N = 5$			$N = 6$			$N = 9$	
Thyroid disease		$N = 12$			$N = 10$			$N = 11$	
Hypercholesterinaemia		$N = 36$			$N = 32$			$N = 27$	
Osteoarthritis		$N = 27$			$N = 31$			$N = 34$	

Number of patients is shown as total number, mean values are shown ∅ ± SD.

There was no difference in PD specific impairment and in the progress of PD between the groups.

Table 4 shows the percentage of time spent with different tasks. Patients of group A underwent on average 14.9 ± 0.7, patients of group B 14.7 ± 0.5, and patients of group C 14.8 ± 0.7 cognitive training sessions, respectively ($P < 0.85$). The time spent on different tasks was identical in all three groups.

Patients of group B conducted 11.2 ± 0.5 and patients of group C 11.4 ± 0.6 transfer training sessions ($P < 0.9$).

Table 5 shows the percentage of time spent on different tasks of the transfer training, which was identical in both groups.

3.1. Motor Training. Patients had many difficulties to cope with the tasks. They struggled to find strategies to solve the tasks on their own, for example, they had difficulties to find the correct path through the parcours with obstacles and to follow the changing rules (set shifting). Patients tended

Table 4: Distribution of cognitive training.

Training section	Percentage of time spent (%)
Planning strategies	17
Mnemonics	15
Decision making	15
Set shifting	15
Calculation	10
Navigational skills	10
Information speed processing	8
Summary	5
Concentration	5

Table 5: Transfer training: time spent on different tasks.

Tasks	Percentage of time spent (%)
Planning and sequencing	18
Reasoning	11
Concentration	11
Memory	10
Working memory	8
Anticipation	8
Prospective memory	7
Cognitive information speed	7
Social rules	5
Association	5
Cognitive estimation	4

to perseverate. Walking through a room with eyes closed only guided by different touches of the therapist challenged the patients as well since PD-patients have both deficits in proprioception and in perception of stimuli. The type of tasks and exercises were new to the majority of patients. PD-patients needed more time and repeated instructions to learn mental imagery. The lessons were conducted as individual lessons. It was not possible to conduct group lessons. About 40% of the training took place outdoors, 60% in the gym.

3.2. Assessment of the Caregivers. The caregivers' knowledge about Parkinson's disease was assessed with a questionnaire at the end of the training programme. They were able to answer on average 27 ± 2.1 questions out of 30 compared to 20.2 ± 4.5 questions prior to the education programme. The carers' burden scale did not reveal any significant differences compared to baseline assessment. However, the questionnaire showed low burden in 60% of carers of group A, in 58% of group B, and in 62% of group C. Moderate burden was revealed in 30% of carers in group A, in 33% in group B, and 32% in group C. However, caregivers reported in a semi structured interview at the end of the education programme that they felt more confident. There was no difference between the caregivers of group A, B, and C.

3.3. Training at Home. 60% of patients of group A continued practising cognitive tasks 3 times a week for 45 min, while 40% conducted the training only once or twice per week. All patients of group B tried to continue the transfer tasks learnt during the rehabilitation, but further assessment showed that only 60% performed transfer tasks following a regular schedule. 75% of the patients of group B practised cognitive tasks 3 times a week. 90% of patients of group C pursued the training at home with the same quantity and intensity. They conducted a motor training three times a week, most often accompanied by their partners. The partners of patients of groups B and C managed to support their patients in practising transfer tasks. They asked them to prepare meals, to write the shopping list, or to go to the bank.

3.4. Results of Neuropsychological Testing. The screening tests for cognitive functions did not reveal any differences between the groups and the results did exclude dementia. The neuropsychological baseline assessment did not reveal any differences between the groups either. Patients of all three groups had shown deficits mainly in tests addressing executive functions. Consecutively, the performance of the patients was worse on the subtests of the SCOPA-COG, semantic fluency, LURIA, dice and assembly pattern, zoo test of the BADS and PASAT. The memory tasks such as immediate and delayed recall were only mildly disturbed.

The multiple-choice word test (MWT-B) was conducted as a measure for premorbid intelligence; the groups did not differ significantly either ($P = 0.78$). Thus, the randomisation process was successful. Table 6 shows the neuropsychological test results over the course of the study.

3.5. Primary Outcome Measure

3.5.1. ADAS-COG. All groups improved on the ADAS-COG significantly shown by a significant linear trend ($F_{\text{lin}}[1, 220] = 150$; $P < 0.001$). Group C improved most indicated by a significant interaction between groups and assessments ($F_{\text{groups}\times\text{assessments}}[1, 220] = 27.26$; $P < 0.001$) and a significant group difference ($F[2, 220] = 7.7$, $P < 0.001$). Further analysis showed that 78% of the patients showed some improvement at the second assessment, 51% of patients of group A, 85% of patients of group B, and 96% of patients of group C. 50% of the patients reached a reduction of the ADAS-COG score of 3 or more points, 18% of group A, 54% of group B, and 76% of group C. Six months after discharge of the rehabilitation unit 35% of patients (50% of patients of group A, 31% of patients of group B, and 28% of group C) showed a deterioration compared to the assessment at the end of the in-patient training programme. Further improvement was observed in 21% patients of group A, 37% patients of group B, and 50% patients of group C.

3.5.2. SCOPA-COG. In accordance the SCOPA-COG test showed a significant difference between the groups (Figure 2). All groups improved, indicated by the linear trend of days ($F_{\text{lin}}[1, 220) = 46.09$; $P < 0.001$). Group C improved most resulting in a significant difference between the groups ($F[2, 220] = 31.4$, df = 2; $P < 0.001$). Since the slopes of the improvements differed between the groups,

Table 6: Summary of neuropsychological test results.

Test	Baseline	T1	T2	Significant differences between the groups
ADAS-Cog	∅ ± SD	∅ ± SD	∅ ± SD	
Group A	21.51 ± 2.27	20.81 ± 2.77	20.5 ± 3.6	
Group B	21.37 ± 4.11	18.33 ± 3.67	18.5 ± 4.2	$P < 0.001$
Group C	22.92 ± 4.02	17.98 ± 2.76	17.4 ± 2.5	
SCOPA-Cog				
Group A	29.07 ± 3.8	27.21 ± 3.6	26.86 ± 3.32	
Group B	29.68 ± 2.87	31.32 ± 3.24	30.71 ± 2.9	$P < 0.001$
Group C	31.83 ± 3.21	39.15 ± 2.9	39.29 ± 2.72	
BADS Zoo (profile)	∅ ± SD	∅ ± SD	∅ ± SD	
Group A	2.5 ± 0.95	3.0 ± 1.2	2.4 ± 1.2	T1: Chi-square: 49.31; $P < 0.001$ T2: Chi-square: 14.42; $P < 0.001$
Group B	2.4 ± 0.9	2.8 ± 1.1	2.6 ± 1.1	
Group C	2.6 ± 0.98	3.54 ± 0.82	3.43 ± 1.0	
BADS instruction	∅ ± SD	∅ ± SD	∅ ± SD	
Group A	2.8 ± 1.3	3.3 ± 1.1	2.9 ± 0.8	T1: Chi-square: 7.1; $P < 0.03$ T2: Chi-square: 9.1; $P < 0.01$
Group B	2.6 ± 1.3	2.9 ± 1.2	3.2 ± 1.1	
Group C	2.7 ± 1.1	3.5 ± 1.1	3.8 ± 0.9	
BADS 6 elements	∅ ± SD	∅ ± SD	∅ ± SD	
Group A	2.8 ± 1.2	3.14 ± 0.89	3.1 ± 0.9	T1: Chi-square: 39.4; $P < 0.001$ T2: Chi-square: 25.3 $P < 0.01$
Group B	2.9 ± 1.2	3.0 ± 1.2	2.9 ± 1.1	
Group C	3.0 ± 0.7	3.55 ± 0.8	3.6 ± 0.9	
PASAT	∅ ± SD	∅ ± SD	∅ ± SD	
Group A	29.94 ± 14.32	32.8 ± 14.83	32.5 ± 13.87	
Group B	31.00 ± 13.32	37.43 ± 12.72	39.57 ± 13.65	$P < 0.001$
Group C	30.4 ± 12.98	46.5 ± 11.5	49.2 ± 13.4	

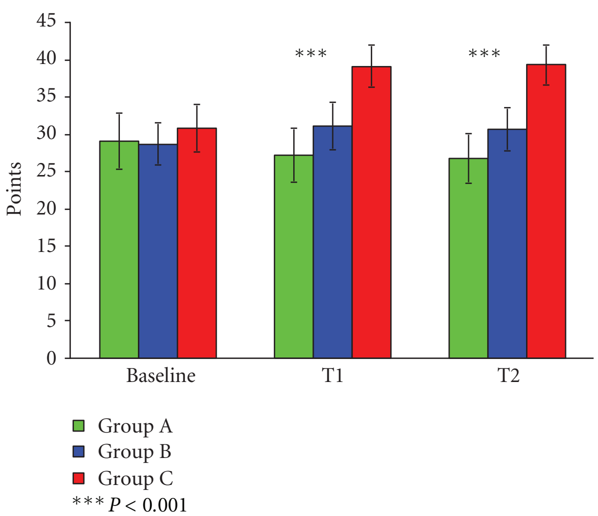


Figure 2: Group C improved significantly more on the SCOPA-COG test than group A and B.

a significant interaction between assessments and groups occurred ($F_{\text{groups}\times\text{assessments}}[2, 220] = 65.63$; $P < 0.001$). Post hoc tests revealed a significant difference between all groups ($P < 0.001$). Patients of group A reached 28.8 ± 3.7 points, group B 30.3 ± 2.7 points, and group C 37.6 ± 3.4 points. After completion of the in-patient training programme 31% of group A, 64% of group B, and 88% of group C had shown a significant improvement on the SCOPA-COG. Six months later at the final assessment 70% of patients of group A, 80% of patients of group B, and 94% of patients of group C had been able to keep their level of performance. Most improvement has been observed in the LURIA, dice, assembly pattern, and MOSAIC test of the SCOPA-COG. The pattern of improvement did not differ between the groups but the percentage of subjects showing an improvement and the speed of recovery.

3.5.3. Executive Function

BADS-Subscales. The BADS-subscales especially the zoo map are a very demanding task requiring excellent planning skills. The subtests of the BADS (rule shift cards, zoo map, modified 6 elements test) showed the following results.

The baseline scores of the rule shift cards did not differ between the groups. There was a mild but significant difference between the groups at the second assessment (Chi-square = 7.1; $P < 0.03$) and final assessment (Chi-square = 9.1; $P < 0.01$).

At baseline assessment group C showed a tendency to perform better on the BADS zoo test. The mean profile scores of all groups were higher at the second assessment, but significant more patients of group C improved compared to groups A and B. There was a clear group difference at the second (Chi-square = 49.31; $P < 0.03$) and third assessment (Chi-square = 14.42; 0.001). At the final assessment six months after the discharge patients of groups A and B had lost most of the previously shown improvement. Only patient of group C managed to keep their level of performance. Thus, group C has been superior to groups B and A immediately after completing the training and at the second assessment six months later.

There was no difference in the performance in the 6 elements test at baseline assessment. Group C showed a greater increase of the average profile scores leading to significant group differences at the second (Chi-square = 39.3; $P < 0.001$) and third assessments (Chi-square = 25.3; $P < 0.001$).

3.5.4. PASAT. The results of the PASAT test did not differ between the groups at baseline assessment, all groups produced on average 50% correct answers. Group A improved only marginally. Groups B and C benefitted from the training programme shown in a significant linear trend for assessments ($F_{\text{lin}}[1, 154] = 63.71$; $P < 0.001$). Since the improvement of the groups differed there was also a significant interaction between assessments and groups ($F_{\text{groups}\times\text{assessments}}[2, 154] = 18.99$; $P < 0.001$). Group C improved significantly more than groups B ($P < 0.03$) and A ($P < 0.001$) ($F[2, 154] = 15.46$; $P < 0.001$).

Only 157 patients (group A: 50, group B: 53, and group C: 54) managed the PASAT test on the first assessment and were included in the statistical model. The other patients did not succeed in finding a strategy to cope with the task. On the second and third assessment 56 patients of group A, 64 patients of group B, and 71 patients of group C scored on the test. Groups B and C showed further improvement between the second and final assessment.

3.5.5. Goal Attainment Scale. The goals were identified at the baseline assessment. On the final assessment it was reviewed whether the goals were obtained. Patients of group C reached more often the main goal than the other groups (Chi-square: 57.1; $P < 0.001$). The detailed analysis of the results is shown in Tables 7 and 8.

The patients had selected the goals based on their self-evaluation of their cognitive impairment and on the advice given by the psychologist after the baseline testing. The main cognitive impairments reported by the patients could be attributed to the following domains: dual tasking, planning of complex and sequential tasks, decision making, rule recognition, rule shifting problems, delayed recall, and difficulties in finding misplaced items.

Table 7 shows the goals patients had chosen and whether they were obtained at the final assessment.

Planning of complex tasks, rule recognition, and shifting and dual tasking were identified as goals most often. While there was no significant difference between the goals chosen by the three groups there was a significant difference between the percentages of patients obtaining the goals between the groups. 71% of patient obtained the goal "decision making," but only 40% of patients of group A.

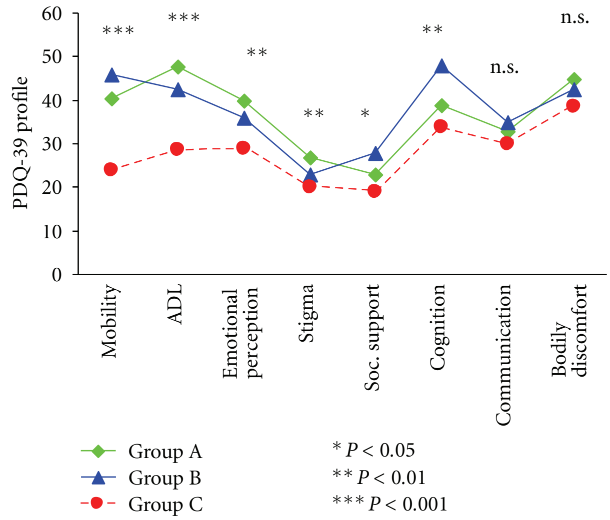


Figure 3: PD-patients of group C reported less PD-specific impairment at the final assessment. The y-axis shows the percentage of the maximal possible sum scores. The x-axis shows the 8 subscales of the PDQ-39. The lines represent the scores the groups have achieved in the 8 subscales.

Table 8 shows the improvement of the patients on the GAS scale with reference to their main goals.

More patients of group A compared to group B and C did not obtain the chosen goal or deteriorated compared to baseline, while 27.6% of patients of group C obtained the goal and 39.4% exceeded the expectations mildly and 7.6% substantially. The difference between the groups was significant (Chi-square = 48.23; $P < 0.001$).

3.6. Assessment of Mental State. 15% of the patients in group A, 20% of group B, and 18% of patients of group C reported to suffer from depression and received medication. The results on the HADS depression scale indicated in 20% of patients of group A and group C, respectively, and in 25% of patients of Group B the presence of a mild to moderate depression. The anxiety level was assessed by using the Hamilton Anxiety Scale and did not differ between the groups.

3.7. PD-Specific Impairment at the Final Assessment. The results of the PDQ-39 at the final assessment showed that patients of group C rated their health-related quality of life higher than the other groups. 13.8% of patients of group A, 38% of patients of group B, and 52% of patients of group C reported less impairment due to PD (Figure 3).

3.8. PD-Specific Functioning. The UPDRS score showed a mild improvement in all groups at the final assessment but there were no significant differences between the groups indicating that the cognitive improvement could not be

Table 7: Individual goals chosen by the patients and percentage of goal achievement.

Groups	Goal	Goals chosen Total number	Goals chosen Percentage (%)	Goals obtained Total number	Goals obtained Percentage (%)	Significance
A $N = 71$	Dual tasking	$N = 15$	21.1	$N = 3$	20	$P < 0.001$
B $N = 75$		$N = 14$	18.7	$N = 9$	64	
C $N = 76$		$N = 15$	20	$N = 10$	67	
A $N = 71$	Planning of complex tasks	$N = 15$	21.2	$N = 3$	20	$P < 0.001$
B $N = 75$		$N = 16$	21.3	$N = 9$	56.3	
C $N = 76$		$N = 17$	22.4	$N = 10$	58.9	
A $N = 71$	Decision making	$N = 10$	14.1	$N = 4$	40	$P < 0.001$
B $N = 75$		$N = 11$	14.7	$N = 6$	54.5	
C $N = 76$		$N = 14$	18.4	$N = 10$	71.4	
A $N = 71$	Rule recognition and rule shifting	$N = 13$	18.3	$N = 4$	30.8	$P < 0.01$
B $N = 75$		$N = 16$	21.3	$N = 7$	43.8	
C $N = 76$		$N = 14$	18.4	$N = 10$	71.4	
A $N = 71$	Delayed recall	$N = 12$	16.9	$N = 3$	25	$P < 0.001$
B $N = 75$		$N = 12$	16	$N = 7$	58.3	
C $N = 76$		$N = 11$	14.5	$N = 9$	82	
A $N = 71$	Search strategies	$N = 6$	8.5	$N = 4$	67	$P < 0.01$
B $N = 75$		$N = 6$	8	$N = 5$	83.3	
C $N = 76$		$N = 5$	6.6	$N = 4$	80	

Table 8: Effect of the training programme on the main goals (GAS).

GAS	Group A $N = 71$ Total	Group A Percent	Group B $N = 75$ Total	Group B Percent	Group C $N = 76$ Total	Group C Percent	Total Total	Total Percent
−3	$N = 12$	16.7	$N = 6$	8	$N = 2$	2.6	$N = 20$	8.9
−2	$N = 10$	13.8	$N = 5$	6.7	$N = 3$	3.9	$N = 18$	23.7
−1	$N = 28$	40.3	$N = 21$	28	$N = 18$	23.6	$N = 67$	30
0	$N = 13$	18.1	$N = 19$	25.3	$N = 23$	30.2	$N = 55$	24.7
1	$N = 8$	11.1	$N = 24$	32	$N = 24$	26.3	$N = 56$	25.1
2	$N = 0$	0	$N = 0$	0	$N = 6$	7.9	$N = 6$	2.7
	71		75		76		223	

−3 = worse than start of treatment, −2 = no change, −1 = some improvement, 0 = goal achievement, +1 = slight over-achievement, +2 = great over-achievement.

Table 9: UPDRS.

	Group A $N = 72$	Group B $N = 75$	Group C $N = 76$
Baseline			
UPDRS Motor scale	38.56 ± 12.44	37.53 ± 10.76	38.4 ± 11.78
UPDRS Sum Score	59.20 ± 12.4	60.3 ± 12.4	61.5 ± 12.8
Final assessment			
UPDRS Motor scale	34.1 ± 11.4	34.2 ± 11.2	35.2 ± 12.4
UPDRS Sum Score	55.4 ± 12.4	56.3 ± 11.5	57.2 ± 11.4

referred to a nonspecific effect resulting from general physical improvement (Table 9).

Performance in Activities of Daily Living at the Final Assessment and Evaluation of the Training Programme by Patients and Caregivers. The patients of group C reported that they had adapted a more active life style, felt more confident in activities of daily living, and had taken over some more chores. They perceived their partners and caregivers as being helpful. They enjoyed the participation of their partners in conjoint sports activities.

Patients of group B also regarded the training programme as helpful but reported of having still problems with activities of daily living. Patients of group A had more difficulties with transfer of skills into daily life and the carryover effect was smaller than in the other groups.

65% of the caregivers of patients in group C, 54% of caregivers of group B, and 49% of caregivers of group A found that competence and cognition of their patients had improved in activities of daily living. A deterioration of the performance in daily living was reported in 11% of group C, 17% of group B, and 25% of group A.

Caregivers felt more relaxed and competent to manage difficult situations, while patients accepted the guidance of their caregivers better than prior to the training. Caregivers felt confident to support the partners with the training at home.

Patients of group A felt that the cognitive training was arduous at times. Some patients perceived the training as stressful. Patients of groups B and C were asked to compare the training programmes. Patients of group C preferred the motor training to transfer training and cognitive pencil and paper tasks. 80% of patients judged the training as strenuous and felt sometimes exhausted. 30% of patients reported of being frustrated at times but did not ask for help or further explanations.

In summary patients who conducted a multimodal cognitive rehabilitation programme improved most on the ADAS-COG and SCOPA-COG, reported a better quality of life, were more active, and continued coping with daily tasks. Patients of group C were physically more active after the training programme and a higher percentage of patients of group C continued with the cognitive training. Physical improvement did not explain the difference in cognitive performance after the end of the study.

4. Discussion

Dementia is a part of the Parkinson's disease spectrum. Currently, there is no treatment aimed at halting or reversing disease progression [6]. Most treatment approaches are based on substituting neurotransmitter deficits. However, the effect of the drug treatment is limited and inconsistent among PD-patients. Therefore, other treatment options are needed. The current study has shown that a multimodal cognitive training programme might improve cognitive performance in PD-patients with mild cognitive impairment. The effect of the multimodal cognitive training on cognitive functioning was comparable to the effect found by Emre et al. in the rivastigmine trial [6]. The superiority of group C with respect to the cognitive assessments suggests that a diversified and challenging training is more effective. The results support our hypothesis that a special motor training might positively affect cognitive functioning. In children movement skill training is used to improve working memory, inhibition of impulses, attention, and visuospatial capacity. The expression psychomotor training is often used in Germany for physical training that combines physical and cognitive tasks. Oswald et al. [46, 47] were the first who called the physical training conducted in the SIMA project psychomotor training. They found that a combined psychomotor and memory training led to an improvement of psychomotor performance and to an improvement of cognitive performance and competence. Neither the psychomotor training nor the memory training on its own resulted in such effects. Oswald et al. [46, 47] assumed that neurophysiological changes led to a provision of reserve capacity of CNS performance. Therefore, emphasis should be placed on the reduction of cognitive load in neuropsychological training programmes. Goebel et al. [48] postulated as well to reduce instructions and working memory load during the training and to use procedures which lead to a more automated, implicit strategy application which demand less executive control [49–51].

Oswald et al. [46, 47] also suggested that the memory training affects psychomotor performance confirming the "memory-movement-hypothesis." An interaction between motor training and memory training might support remembering movement sequences. Our results confirm the hypothesis of Oswald et al. [46] that more diverse training programme enhances motivation and resulted in more regular training. Patients of group C exercised more physically and conducted more paper and pencil lesions at home than the other groups indicating that the attitude towards learning was positively influenced. In addition, the partners of patients in group C participated actively in the sport programme which helped patients to adhere to the training programme. As known from questionnaires we had sent to patients and their families inquiring about the training; social aspects are very important for PD-patients. Home-based multimodal cognitive training programme was sufficient to keep the performance of the second assessment in patients of group C. The psychomotor training combined with endurance training might have contributed to the superiority of group C. There is a huge body of literature suggesting a prevention of cognitive decline by life long exercise or even an improvement of cognitive deficits by physical activity. Executive functions may be selectively maintained or improved in people with better physical condition provided by physical training [52]. The importance of aerobic physical exercise on cognitive functions, especially on executive functions, has been shown [19–21, 26]. The studies have been mainly conducted in healthy elderly or patients with dementia but older people with PD can benefit their executive functions in the same way, as do their peers without PD. The results of some studies have shown that brain areas undergoing biological aging benefit most from endurance sports. Even structural changes have been observed [21]. Since group C conducted more training lessons at home compared to the other groups, one might argue that the superiority of group C was rather due to the quantity of the training than to the content. However, group C has already performed better at the second assessment and group A had shown poorer results than groups B and C. At this time all groups spent a similar amount of time with training and received similar attention by the therapists. Thus, the content of the training might be responsible for the different performance of the groups. The superiority of group B compared to group A suggests the efficacy of the transfer tasks. The psychomotor training helped group C to improve further, especially in the challenging executive tasks regarding rule cognition, set shifting, and decision making. The authors had also taken care during the study design that groups B and A received relaxation training and physiotherapy as compensation for the motor training of group C during the 6-month training period at home. These treatment offers were also accepted by the patients but especially patients of group A did not practise cognitive lessons at home as much as they were advised to do. Home-based cognitive training without transfer and physical training as performed by group A was less attractive for the patients.

The groups managed the executive tests very differently. The BADS zoo map is a very challenging test as mentioned above and requires various training approaches to achieve an improvement. Only patients of group C obtained a permanent improvement on this test.

Thus, the different training schedules affect the training outcome also regarding the type of tasks, which were better performed. Patients of group C managed executive tasks better than group A and B, which is shown by the better performance on the BADS test battery and the better results on the SCOPA-COG. Exercise and endurance training is thought to improve executive functions most. Furthermore, brain areas which are more vulnerable for age-related volume lose benefit most from physical exercise.

Patients with mild cognitive impairment benefit more from cognitive training than more advanced patients. Studies have shown that the effect of physical exercise on demented patients is limited. Patients have to be capable of understanding the training programme. Depression might also influence the performance in neuropsychological tests. Klepac et al. [53] have found that depression preceding PD motor signs might favour poorer cognitive abilities. However, there was no significant difference between the groups regarding the percentage of patients being depressed and the severity of depression. Thus, depression and anxiety were not confounding factors and not responsible for the different treatment outcomes of the three groups. Assessment bias in favour for one treatment can be excluded because the movement disorder specialists conducting the tests were blinded to the treatment arms.

The findings of the study confirmed as well that PD-patients benefit from a specific cognitive training. Based on the results of a previous study [54], we had chosen an individual training approach. Therefore, patients did not undergo a standardised cognitive training programme but a programme tailored to their needs. Interestingly, the results of the baseline testing and the psychologist's training suggestions were very often concordant with the requests of the patients. Therefore, the goal attainment scale was very suitable to represent both the training suggestions and the patients' requests. The fact that more patients of group C attained the selected goals might also support the strength of a multimodal training. Resembling the results of the executive tests some goals seemed to be more difficult to attain (see Tables 7 and 8). Patients struggled more to attain goals such as rule generation or rule shifting while dual tasking and memory improvement were easier to accomplish. However, group C was also more successful in achieving an improvement in planning of complex tasks, rule generation, rule shifting, and decision finding than groups B and C.

In contrast to a study by París et al. [55], the present study suggested a translation of improved cognitive performance into daily living. The patients' caregivers also reported an improved competence in real life. However, patients practised during the transfer training situations of everyday life. Hence, they did not face completely new situations in their daily life. París et al. [55] found an improvement in attention, information processing speed, memory, visuospatial and visuo-constructive abilities, semantic verbal fluency, and executive functions but no improvement of cognitive difficulties in daily living.

Sinforiani et al. [56] observed a carryover effect after completion of a 6-week cognitive rehabilitation training including cognitive and physical training. The authors suggested that the combined cognitive rehabilitation training exerts its positive effects by reinforcing cognitive strategies with improvement of frontal lobe functions.

Quality of life is closely associated with nonmotor symptoms of PD, especially cognitive function [8]. Consequentially Group C scored much higher on the PDQ-39 than the other groups. Thus, health-related quality of life was improved markedly in these patients. In contrast to our results París et al. [55] did not find any benefits of the cognitive training in self-reported quality of life. Probably, the cognitive training has to lead to improvements in daily living in order to improve quality of life.

Research over the last decade has shown that cognitive deficits affect motor performance. Patients with cognitive deficits had more difficulties in motor tests than patients without cognitive deficits. Hausdorff et al. [57] have found a close correlation between walking and executive functions. Yogev et al. [58] have shown that gait variability in dual tasking is closely associated with the performance in executive tasks. The difficulties that patients of group C experienced while solving the motor tasks have been in accordance with these results. Nevertheless, most of the patients managed to cope with the training programme, though some patients felt overstrained and quitted the programme.

4.1. Limitations of the Study. One might criticise that we compared three different treatment arms and did not include a control group without cognitive training in this study. However, patients were enrolled into the study during their stay on a rehabilitation unit and complained of a deterioration of their cognitive performance. For this reason, it was not possible to withhold treatment. Further, we had shown the superiority of a cognitive training compared to standard treatment in a previous study [54].

Another limitation is that there are no evidence-based data for the transfer training. Further research is necessary to evaluate and validate which transfer exercises are useful tools. The psychomotor training or movement skill training has been used for many years in children and has been applied in patients with dementia [53, 54]. However, it has not been validated in PD-patients so far. The selection of tasks has been based on the experience of the therapists and medical staff and published data which were based on the work with children.

One might also argue which improvement on the ADAS-COG or SCOPA-COG might be clinically relevant. However, the scales are validated and had been often applied in clinical trials. The clinical relevance of the improvements has also been shown by the observed translation into real life. Furthermore, due to the short follow-up period of 6 months we cannot report on long-term effects. However, studies assessing long-term results are very difficult to conduct since it is very difficult to keep the medication stable.

4.2. Strengths of the Study. To our knowledge, the results of the current study show for the first time, that a multimodal cognitive training in patients with Parkinson's disease can lead to improvements of cognitive function and improves quality of life. In addition, there was some translation of the cognitive improvement into real life. We also want to emphasize that it was a blinded randomised study and the drop-out rate was low.

5. Conclusion

In conclusion, we have shown that PD-patients with cognitive deficits benefit from a multimodal cognitive training. The multimodal training was superior to a paper- and pencil-based cognitive training. The combination of the cognitive training with a motor training seems to be most successful and the short-term effect of the training on cognitve functions was comparable to the effect found in the rivastigmine trial. However, we cannot predict the long-term effect of the cognitive multimodal training. The study has shown some translation of cognitive improvements into "real" life. Admittedly, the multimodal training of cognitive functions is time consuming, requires high motivation of the patients, and put demands on resources. Due to the quantity and quality of the trainings sessions it will also be costly. On the other hand dementia is a risk factor for falls, high morbidity and transfer to nursing homes, which increases the costs for the patients' care substantially and jeopardizes the patients' quality of life.

Acknowledgment

The authors would like to thank Dr. Werner Jackstädt-Stiftung for the generous support of the study.

References

[1] R. C. Chen, S. F. Chang, C. L. Su et al., "Prevalence, incidence, and mortality of PD: a door-to-door survey in Ilan County, Taiwan," *Neurology*, vol. 57, no. 9, pp. 1679–1686, 2001.

[2] A. Schrag, Y. Ben-Shlomo, and N. P. Quinn, "Cross sectional prevalence survey of idiopathic Parkinson's disease and parkinsonism in London," *British Medical Journal*, vol. 321, no. 7252, pp. 21–22, 2000.

[3] J. Douglas, D. J. Gelb, E. Oliver, and S. Gilman, "Diagnostic criteria for Parkinson disease," *Archives of Neurology*, vol. 56, no. 1, pp. 33–39, 1999.

[4] A. J. Hughes, S. E. Daniel, L. Kilford, and A. J. Lees, "Accuracy of clinical diagnosis of idiopathic Parkinson's disease: a clinico-pathological study of 100 cases," *Journal of Neurology Neurosurgery and Psychiatry*, vol. 55, no. 3, pp. 181–184, 1992.

[5] D. Aarsland, K. Andersen, J. P. Larsen, A. Lolk, and P. Kragh-Sørensen, "Prevalence and characteristics of dementia in Parkinson disease: an 8-year prospective study," *Archives of Neurology*, vol. 60, no. 3, pp. 387–392, 2003.

[6] M. Emre, D. Aarsland, A. Albanese et al., "Rivastigmine for dementia associated with Parkinson's disease," *New England Journal of Medicine*, vol. 351, no. 24, pp. 2509–2518, 2004.

[7] G. Levy, M. X. Tang, E. D. Louis et al., "The association of incident dementia with mortality in PD," *Neurology*, vol. 59, no. 11, pp. 1708–1713, 2002.

[8] A. Schrag, M. Jahanshahi, and N. Quinn, "What contributes to quality of life in patients with Parkinson's disease?" *Journal of Neurology Neurosurgery and Psychiatry*, vol. 69, no. 3, pp. 308–312, 2000.

[9] D. Aarsland, J. P. Larsen, K. Karlsen, N. G. Lim, and E. Tandberg, "Mental symptoms in Parkinson's disease are important contributors to caregiver distress," *International Journal of Geriatric Psychiatry*, vol. 14, no. 10, pp. 866–874, 1999.

[10] D. Aarsland, J. P. Larsen, E. Tandberg, and K. Laake, "Predictors of nursing home placement in Parkinson's disease: a population-based, prospective study," *Journal of the American Geriatrics Society*, vol. 48, no. 8, pp. 938–942, 2000.

[11] M. Emre, "Dementia associated with Parkinson's disease," *Lancet Neurology*, vol. 2, no. 4, pp. 229–237, 2003.

[12] H. Braak, K. Del Tredici, U. Rüb, R. A. I. De Vos, E. N. H. Jansen Steur, and E. Braak, "Staging of brain pathology related to sporadic Parkinson's disease," *Neurobiology of Aging*, vol. 24, no. 2, pp. 197–211, 2003.

[13] E. K. Perry, M. Curtis, and D. J. Dick, "Cholinergic correlates of cognitive impairment in Parkinson's disease: comparisons with Alzheimer's disease," *Journal of Neurology Neurosurgery and Psychiatry*, vol. 48, no. 5, pp. 413–421, 1985.

[14] I. Litvan, D. Aarsland, C. H. Adler et al., "MDS task force on mild cognitive impairment in Parkinson's disease: critical review of PD-MCI," *Movement Disorders*, 2011.

[15] E. E. Smith and J. Jonides, "Storage and executive processes in the frontal lobes," *Science*, vol. 283, no. 5408, pp. 1657–1661, 1999.

[16] P. W. Burgess and T. Shallice, "Response suppression, initiation and strategy use following frontal lobe lesions," *Neuropsychologia*, vol. 34, no. 4, pp. 263–272, 1996.

[17] E. K. Miller and J. D. Cohen, "An integrative theory of prefrontal cortex function," *Annual Review of Neuroscience*, vol. 24, pp. 167–202, 2001.

[18] R. D. Abbott, L. R. White, G. W. Ross, K. H. Masaki, J. D. Curb, and H. Petrovitch, "Walking and dementia in physically capable elderly men," *Journal of the American Medical Association*, vol. 292, no. 12, pp. 1447–1453, 2004.

[19] S. Colcombe and A. F. Kramer, "Fitness effects on the cognitive function of older adults: a meta-analytic study," *Psychological Science*, vol. 14, no. 2, pp. 125–130, 2003.

[20] S. J. Colcombe, K. I. Erickson, N. Raz et al., "Aerobic fitness reduces brain tissue loss in aging humans," *Journals of Gerontology A*, vol. 58, no. 2, pp. 176–180, 2003.

[21] S. J. Colcombe, K. I. Erickson, P. E. Scalf et al., "Aerobic exercise training increases brain volume in aging humans," *Journals of Gerontology A*, vol. 61, no. 11, pp. 1166–1170, 2006.

[22] D. Laurin, R. Verreault, J. Lindsay, K. MacPherson, and K. Rockwood, "Physical activity and risk of cognitive impairment and dementia in elderly persons," *Archives of Neurology*, vol. 58, no. 3, pp. 498–504, 2001.

[23] Y. Rolland, G. Abellan van Kan, and B. Vellas, "Healthy brain aging: role of exercise and physical activity," *Clinics in Geriatric Medicine*, vol. 26, no. 1, pp. 75–87, 2010.

[24] G. Nelles, "Cortical reorganization—effects of intensive therapy: results from prospective functional imaging studies," *Restorative Neurology and Neuroscience*, vol. 22, no. 3-4, pp. 239–244, 2004.

[25] R. B. Shepherd, "Exercise and training to optimize functional motor performance in stroke: driving neural reorganization?" *Neural Plasticity*, vol. 8, no. 1-2, pp. 121–129, 2001.

[26] A. F. Kramer, S. Hahn, N. J. Cohen et al., "Ageing, fitness and neurocognitive function," *Nature*, vol. 400, no. 6743, pp. 418–419, 1999.

[27] J. Baatile, W. E. Langbein, F. Weaver, C. Maloney, and M. B. Jost, "Effect of exercise on perceived quality of life of individuals with Parkinson's disease," *Journal of Rehabilitation Research and Development*, vol. 37, no. 5, pp. 529–534, 2000.

[28] I. Reuter, M. Engelhardt, K. Stecker, and H. Baas, "Therapeutic value of exercise training in Parkinson's disease," *Medicine and Science in Sports and Exercise*, vol. 31, no. 11, pp. 1544–1549, 1999.

[29] K. Dujardin, A. Duhamel, M. Delliaux, C. Thomas-Antérion, A. Destée, and L. Defebvre, "Cognitive complaints in Parkinson's disease: its relationship with objective cognitive decline," *Journal of Neurology*, vol. 257, no. 1, pp. 79–84, 2010.

[30] S. Fahn, R. L. Elton, and The Members of the UPDRS Development Committee, "Unified Parkinson's disease rating scale," in *Recent Developments in Parkinson's Disease II*, S. Fahn, C. D. Marsden, M. Goldstein et al., Eds., pp. 153–163, McMillan, New York, NY, USA, 1987.

[31] Royal College of Physicians and the Intercollegiate Stroke Working Party, *National Clinical Guideline for Stroke*, RCP, London, UK, 3rd edition, 2008.

[32] E. Kalbe, P. Calabrese, N. Kohn et al., "Screening for cognitive deficits in Parkinson's disease with the Parkinson neuropsychometric dementia assessment (PANDA) instrument," *Parkinsonism and Related Disorders*, vol. 14, no. 2, pp. 93–101, 2008.

[33] M. F. Folstein, S. E. Folstein, and P. R. McHugh, "'Mini mental state'. A practical method for grading the cognitive state of patients for the clinician," *Journal of Psychiatric Research*, vol. 12, no. 3, pp. 189–198, 1975.

[34] W. G. Rosen, R. C. Mohs, and K. L. Davis, "A new rating scale for Alzheimer's disease," *American Journal of Psychiatry*, vol. 141, no. 11, pp. 1356–1364, 1984.

[35] S. Talwalker, "Assessment of AD with the ADAS-cog," *Journal of Geriatric Psychiatry and Neurology*, vol. 9, pp. 39–46, 1996.

[36] J. Marinus, M. Visser, N. A. Verwey et al., "Assessment of cognition in Parkinson's disease," *Neurology*, vol. 61, no. 9, pp. 1222–1228, 2003.

[37] D. M. A. Gronwall, "Paced auditory serial addition task: a measure of recovery from concussion," *Perceptual and Motor Skills*, vol. 44, no. 2, pp. 367–373, 1977.

[38] B. A. Wilson, J. J. Evans, H. Emslie, N. Alderman, and P. Burgess, "The development of an ecologically valid test for assessing patients with a dysexecutive syndrome," *Neuropsychological Rehabilitation*, vol. 8, no. 3, pp. 213–228, 1998.

[39] S. Lehrl, *Mehrfach-Wortschatz-Intelligenztest: MWT-B*, Perimed Fachbuch-Verlagsgesellschaft mbH, Erlangen, Germany, 1989.

[40] A. S. Zigmond and R. P. Snaith, "The hospital anxiety and depression scale," *Acta Psychiatrica Scandinavica*, vol. 67, no. 6, pp. 361–370, 1983.

[41] C. Jenkinson, V. Peto, R. Fitzpatrick, R. Greenhall, and N. Hyman, "Self-reported functioning and well-being in patients with Parkinson's disease: comparison of the short-form wealth survey (SF-36) and the Parkinson's disease questionnaire (PDQ-39)," *Age and Ageing*, vol. 24, no. 6, pp. 505–509, 1995.

[42] S. J. G. Lewis, A. Slabosz, T. W. Robbins, R. A. Barker, and A. M. Owen, "Dopaminergic basis for deficits in working memory but not attentional set-shifting in Parkinson's disease," *Neuropsychologia*, vol. 43, no. 6, pp. 823–832, 2005.

[43] N. Fournet, O. Moreaud, J. L. Roulin, B. Naegele, and J. Pellat, "Working memory functioning in medicated Parkinson's disease patients and the effect of withdrawal of dopaminergic medication," *Neuropsychology*, vol. 14, no. 2, pp. 247–253, 2000.

[44] S. Golubović, T. Tubić, and S. Marković, "Psycho-motor re-education-movement as therapeutic method," *Medicinski Pregled*, vol. 64, no. 1-2, pp. 61–63, 2011.

[45] T. Golubović, J. Maksimović, B. Golubović, and N. Glumbić, "Effects of exercise on physical fitness in children with intellectual disability," *Research in Developmental Disabilities*, vol. 33, no. 2, pp. 608–614, 2012.

[46] W. D. Oswald, R. Rupprecht, T. Gunzelmann, and K. Tritt, "The SIMA-project: effects of 1 year cognitive and psychomotor training on cognitive abilities of the elderly," *Behavioural Brain Research*, vol. 78, no. 1, pp. 67–72, 1996.

[47] W. D. Oswald, T. Gunzelmann, R. Rupprecht, and B. Hagen, "Differential effects of single versus combined cognitive and physical training with older adults: the SimA study in a 5-year perspective," *European Journal of Ageing*, vol. 3, no. 4, pp. 179–192, 2006.

[48] S. Goebel, H. M. Mehdorn, and B. Leplow, "Strategy instruction in Parkinson's disease: influence on cognitive performance," *Neuropsychologia*, vol. 48, no. 2, pp. 574–580, 2010.

[49] A. D. Baddeley, *Working Memory*, Clarendon Press, Oxford, UK, 1986.

[50] D. A. Norman and T. Shallice, "(1980) Attention to action: willed and automatic control of behaviour," in *Cognitive Neuroscience A*, M. S. Gazzaniga, Ed., Blackwell, Oxford, UK, 2000.

[51] G. Sammer, I. Reuter, K. Hullmann, M. Kaps, and D. Vaitl, "Training of executive functions in Parkinson's disease," *Journal of the Neurological Sciences*, vol. 248, no. 1-2, pp. 115–119, 2006.

[52] J. D. Churchill, R. Galvez, S. Colcombe, R. A. Swain, A. F. Kramer, and W. T. Greenough, "Exercise, experience and the aging brain," *Neurobiology of Aging*, vol. 23, no. 5, pp. 941–955, 2002.

[53] N. Klepac, S. HajnÅek, and V. Trkulja, "Cognitive performance in nondemented nonpsychotic parkinson disease patients with or without a history of depression prior to the onset of motor symptoms," *Journal of Geriatric Psychiatry and Neurology*, vol. 23, no. 1, pp. 15–26, 2010.

[54] K. Hullmann, G. Sammer, and I. Reuter, "Training of executive functions in Parkinson's disease," in *Medimont International Proceedings*, pp. 143–148, 2004.

[55] A. P. París, H. G. Saleta, M. de la Cruz Crespo Maraver et al., "Blind randomized controlled study of the efficacy of cognitive training in Parkinson's disease," *Movement Disorders*, vol. 26, no. 7, pp. 1251–1258, 2011.

[56] E. Sinforiani, L. Banchieri, C. Zucchella, C. Pacchetti, and G. Sandrini, "Cognitive rehabilitation in Parkinson's disease.," *Archives of Gerontology and Geriatrics*, supplement 9, pp. 387–391, 2004.

[57] J. M. Hausdorff, G. Yogev, S. Springer, E. S. Simon, and N. Giladi, "Walking is more like catching than tapping: gait in the elderly as a complex cognitive task," *Experimental Brain Research*, vol. 164, no. 4, pp. 541–548, 2005.

[58] G. Yogev, N. Giladi, C. Peretz, S. Springer, E. S. Simon, and J. M. Hausdorff, "Dual tasking, gait rhythmicity, and Parkinson's disease: which aspects of gait are attention demanding?" *European Journal of Neuroscience*, vol. 22, no. 5, pp. 1248–1256, 2005.

Hindawi Publishing Corporation
Journal of Aging Research
Volume 2012, Article ID 890679, 8 pages
doi:10.1155/2012/890679

Research Article

Barriers Are Not the Limiting Factor to Participation in Physical Activity in Canadian Seniors

Kristy L. Smith, Kelly Carr, Alexandra Wiseman, Kelly Calhoun, Nancy H. McNevin, and Patricia L. Weir

Department of Kinesiology, University of Windsor, Windsor, ON, Canada N9B 3P4

Correspondence should be addressed to Patricia L. Weir, weir1@uwindsor.ca

Received 16 March 2012; Revised 8 July 2012; Accepted 19 July 2012

Academic Editor: Lynn Rochester

The identification of barriers to physical activi[illegible]d for many decades to explain exercise behavior in older adults. Typically health concerns are the num[illegible]tion. Data from CCHS-HA dataset ($N = 20{,}875$) were used to generate a sample of Canadians, 60+ y[illegible]health condition limitation, illness, or injury as a barrier to participation in physical activity ($n = 4{,}9$[illegible]ique in terms of the study of barriers to participation. While the vast majority of older adults partici[illegible]% did not. The relationships between nonparticipation, barriers, self-reported health status, and chro[illegible] determined using binary logistic regression. The main findings suggest that traditional barriers and s[illegible] not responsible for nonparticipation. Nonparticipation was best predicted by chronic health conditions suggesting a disconnect between self-reported health status and underlying health conditions. The data are clear in suggesting that barriers are not the limiting factor and physical activity programming must be focused on meeting the health needs of our aging population.

1. Introduction

The world's population is aging. In 2009, 14% of Canada's 32 million people were aged 65 years or older. This proportion is expected to rise to between 23 and 25% by 2036; effectively doubling the number of seniors observed in 2009 [1], placing increased demand on the health care system and the nation's workforce. With this dramatic demographical change in the population, it has become increasingly important to find effective ways to improve health and prolong independence among older adults. Participation in regular physical activity can provide numerous physiological, cognitive, and psychological health benefits in the aging population. While some level of decline, such as reduced muscle strength and slower responding, is a normal part of the aging process [2], there is evidence that habitual exercise can minimize the physiological effects of an otherwise sedentary lifestyle and prolong active life expectancy [3]. However, despite this knowledge, many individuals do not engage in recommended levels of regular physical activity [4], and the Seniors in Canada report card [5] gave Canadian seniors a grade of C+ for participation in physical activity, with 62% of seniors being inactive.

The sizeable proportion of inactive individuals forces a critical examination of the unique challenges faced by older adults, aimed at developing the most effective health strategies to promote physical activity in this cohort [6]. Efforts to optimize health in this manner have led to numerous studies focused on identifying barriers and motivators to exercise among the older adult population, and several determinants have been consistently identified in the research literature as significant factors related to exercise behaviour among older individuals [7]. Considerations such as health concerns [6, 8, 9], lack of knowledge [6, 10], fear of falling or injury [6, 9, 11], time constraints [6, 8, 9], and lack of transportation [6] are generally reported as disincentives to participation by older adults.

Typically, issues contributing to the lack of participation are elicited through the use of focus group discussions [6, 9], interviews [11], or questionnaires [12]. These studies can be complex, such that some issues such as health problems are highly related to each other and can function both as a barrier and a motivator to exercise [6, 8]. In addition, the aging population is a diverse group [9, 13], with no one common barrier reported across all racial and ethnic groups [9]. Level of mobility [12], current exercise status [9], and past experiences [11] can all influence what respondents identify as posing a barrier or an enabler to physical activity.

The majority of older adults do, however, identify at least one of these considerations. In the cohort studied by O'Neill and Reid [14], 87% of the elderly respondents identified at least one barrier that prevents them from engaging in exercise. While the most common barriers appear to be related to health concerns, there are members of the sedentary population that do not identify any barriers to physical activity participation. For example, Cohen-Mansfield et al. [8] stated that 89 of their 324 person sample either reported no barriers at all or that they already engaged in exercise. Ironically, Connell et al. [15] reported that good health itself was a barrier to exercise because it provided the older adults with no reason to exercise.

The goal of the current study was to identify the relationship, if any, between what older adults perceived as barriers to physical activity and participation. Because previous research has suggested that health concerns pose a significant barrier to participation and would thus likely skew the data towards health-related barriers, we chose to focus on a subset of older adults who reported having no health condition limitation, illness, or injury that prevented them from participating in physical activity. We further delineated the analyses by gender as it has been well documented that older women tend to report more chronic diseases than men and this difference may impact overall participation rates [16–18]. Using data from the Canadian Communities Health Survey-Healthy Aging, an overall sample size of 4,900 seniors was identified, and, of these, 9.4% reported no participation in physical activity over the last seven days. We examined the relationship between their nonparticipation and barriers to participation, self-reported health, and chronic health conditions, and hypothesized that traditional barriers would not predict nonparticipation and there may be a dissociation between self-reported health status and reported chronic health conditions. The overarching goal of this study was to identify why Canadian seniors who do not report a health limitation do not participate in regular physical activity.

2. Methods

2.1. Canadian Communities Health Survey-Healthy Aging (CCHS-HA). Data from the Canadian Community Health Survey-Healthy Aging (CCHS-HA) (Statistics Canada, [19]) were used for the current study. This cross-sectional survey gathered self-reported data on the "factors, influences and processes that contribute to healthy aging through a multidisciplinary approach focusing on health, social and economic determinants" [19, page 1]. CCHS-HA data were derived through a standardized interview of adults aged 45 years of age and greater from the ten provinces in Canada, between December 2008 and November 2009. Using computer-assisted personal interviewing (CAPI), trained field interviewers conducted 94% of the interviews face-to-face, with the remainder being conducted over the telephone. The objectives of the survey were to (1) better understand the aging process; (2) examine how lifestyle impacts health; (3) use a multidisciplinary approach to examine relationships between healthy aging and geographical, social, demographic, and economic factors; and, (4) provide information on successful aging.

2.2. Participants. For the purposes of our study, we only used responses from the CCHS-HA dataset for people aged 60 years and older ($N = 20{,}875$). Given the interest in identifying barriers to participation, we further limited our sample to those seniors who did not identify a "health condition limitation" or "illness or injury" as barriers to participation. This ensured that we were not examining individuals who did not believe they were physically able to participate. Master weights were applied to the overall dataset such that the weights had a mean of 0. The weighting value for each respondent corresponded to the number of persons in the entire population that the respondent represented. The final weighted sample size was 4,900 participants (23.47% of total N; male: $n = 2183$; female: $n = 2717$). To estimate the average age of participants, the mid-point of each age-range was used (e.g., 60–64 years = 62.5). For the 85+ age range, we used 87.5 years, which may result in a slight underrepresentation. The overall average age of the sample was 68.39 ± 6.91 years.

2.3. Variables of Interest

2.3.1. Participation. This was determined using the derived variable for participation in leisure physical activities. This categorical variable indicated whether the respondent had participated in walking for pleasure or exercise, light sports, moderate sports, strenuous sports, and exercises to increase muscle strength and endurance over the 7 days prior to the interview. It was scored as either a "1" indicating reported participation over the last 7 days, a "2" indicating no participation over the last 7 days, or a "9" indicating at least one required activity had not been responded to (these were excluded from future analyses). To confirm differences in participation, the PASE (Physical Activity for the Elderly Scale) score was also examined [20]. The PASE score combines information on the frequency and duration of participation in leisure, household, and occupational activity over a seven-day period. While it does not differentiate frequency and duration, a higher score indicates greater levels of participation.

2.3.2. Barriers to Participation. Thirteen barriers to participation were listed in the CCHS-HA dataset: cost, transportation problem, not available in area, location not physically accessible, location is too far, health condition limitation, illness or injury, fear of injury, lack of time, lack of energy, lack of motivation, lack of skills or knowledge, other. Similar to the participation measure, these were categorical variables where respondents indicated that "yes" it prevented participation, or "no" it did not prevent participation. Of these thirteen variables, ten were used to predict participation. Health condition limitation, illness or injury were used only to identify the current sample, and "other" was not included as it was not possible to draw a conclusion about illness, or injury from this response. The total number of barriers was derived (range: 0–8 barriers; mean = 1.06 ± 0.72 barriers; median = 1.00) from the number of barriers each participant responded "yes" to.

2.3.3. Self-Reported Health Status. Respondents were asked "In general, would you say your health is" excellent, very good, good, fair, poor. This categorical variable was recoded such that excellent and very good were collapsed together, and fair and poor were collapsed together leaving three levels of self-reported health status.

2.3.4. Chronic Health Conditions. The CCHS-HA included a number of self-reported chronic condition variables that were coded categorically. The following seven were used in the current study: (1) vision function—this variable was derived from five items based on the respondent's ability and/or inability to see well enough to read newsprint and be able and/or unable to recognize a friend on the other side of the street, with and/or without glasses or contact lenses. This resulted in three vision categories: no vision problems, problems corrected by lenses, and problems not corrected by lenses. No vision problems and corrected vision were collapsed together resulting in the two categories of no vision problems and vision problems; (2) heart disease—if the respondents reported having either angina or a heart attack, they were considered to have heart disease; (3) chronic obstructive pulmonary disease (COPD)—if the respondent reported having been diagnosed with chronic bronchitis, emphysema, or COPD, which yielded a broad category used to describe limitations in lung airflow; (4) diabetes; (5) osteoporosis; (6) living with the effects of a stroke—this was used to reflect neurological damage; (7) mobility trouble—this variable was derived from five items based on the respondent's ability to walk a short distance and/or around their neighborhood, with and/or without the assistance of another person and/or walking equipment and/or a wheelchair. This resulted in four mobility categories: no mobility problems, mobility problems—no assistance required; mobility problems—requires wheelchair; mobility problems—requires help/cannot walk. The mobility problems were collapsed together resulting in two mobility categories.

2.4. Data Analyses. Logistic regression techniques were used to examine the relationships between nonparticipation and barriers to participation, self-reported health status, and the seven chronic health conditions. Data were analyzed separately for male and female respondents. Odds ratios and 95% confidence intervals were used to identify the risk of nonparticipation as a function of the predictor variables. Due to the small sample size of nonparticipants ($n = 459$), results associated with a large range in confidence intervals should be interpreted with caution as they may overestimate the practical significance. SPSS 19 was used for all data analyses, with $P < 0.05$.

3. Results

Table 1 reveals that a majority of the Canadian seniors in this sample were participants in physical activity over the last 7 days. While a small percentage of the respondents had not been physically active, this is the group of interest. Given that none of these respondents has identified barriers due to a health condition limitation, illness, or injury, the data show that approximately 10% do not participate in physical activity. Lower participation in the nonparticipants was confirmed through a lower PASE score (139.68) as compared to participants (142.99). Overall, the majority of the respondents report excellent/very good health, and a large majority do not suffer from the identified health conditions.

As can been seen in Table 2, approximately 89% of participants, regardless of participation level, did not identify a barrier. When the barriers were used to predict nonparticipation, the results of the logistic regressions suggested that older males and females have different barriers to participation (see Table 3). Males were more likely to be nonparticipants as a function of the availability of the activity (OR = 4.50, CI = 1.09, 18.68), while females were more likely to be a nonparticipants due to a lack of time (OR = 1.45, CI = 1.09, 1.92). Nonparticipation was not related to a lack of motivation in males (OR = 0.69, CI = 0.48, 0.98) and was not related to a lack of energy in females (OR = 0.54, CI = 0.39, 0.76). Overall, the data suggest that barriers do not appear to be responsible for the nonparticipation of Canadian seniors.

Given that barriers were not strong predictors of nonparticipation, we examined the influence of self-reported general health status. As seen in Table 1, the distribution of respondents across the three levels of health did vary between participants and nonparticipants. Participants more frequently reported their health as being excellent/very good (61.2% versus 49.2%), and fewer participants reported their health as fair/poor (9.1% versus 13.5%). While these differences in frequency existed, for both males and females there was no relationship between self-reported health status and nonparticipation in physical and leisure activities (see Table 4).

With regards to chronic health conditions, males and females identified a different number of predictors to nonparticipation (see Table 5). For males, who identified three predictors, the greatest likelihood of nonparticipation was predicted by vision function (OR = 3.06, CI = 1.30, 7.21), followed by mobility troubles (OR = 2.65, CI = 1.01, 6.95), and diabetes (OR = 1.66, CI = 1.12, 2.44). Women identified four significant predictors of nonparticipation; mobility troubles (OR = 2.71, CI = 1.70, 4.31), COPD (OR = 2.11, CI = 1.34, 3.32), diabetes (OR = 1.85, CI = 1.30, 2.63), and heart disease (OR = 1.54, CI = 1.19, 1.99).

4. Discussion

The purpose of this study was to examine the relationship between barriers to participation, self-reported health status, and chronic health conditions on nonparticipation in physical activity in Canadian seniors aged 60+ years who did not identify a health condition limitation, illness, or injury

Table 1: Sample descriptive statistics (weighted sample).

Variable	Category	*N*	%
Participation	Participant	4438	90.60
	Nonparticipant	459	9.40
PASE	Participant		142.99
	Nonparticipant		139.68
Gender	Male	2180	44.50
	Female	2717	55.50
Self-reported health status	Excellent/very good	2944	60.10
	Participant	2715	61.2
	Nonparticipant	226	49.2
	Good	1491	30.40
	Participant	1320	29.7
	Nonparticipant	171	37.3
	Fair/Poor	465	9.50
	Participant	404	9.1
	Nonparticipant	62	13.5
Chronic health conditions	Vision function		
	Yes	66	1.40
	No	4806	98.60
	Heart disease		
	Yes	2212	45.20
	No	2676	54.80
	COPD		
	Yes	231	4.70
	No	4664	95.30
	Diabetes		
	Yes	595	12.10
	No	4303	87.90
	Osteoporosis		
	Yes	641	13.10
	No	4253	86.90
	Effects of a stroke		
	Yes	63	1.30
	No	4837	98.70
	Mobility trouble		
	Yes	149	3.10
	No	4748	96.90

as a barrier to participation. Across both participants and nonparticipants, respondents on average identified 1.06 ± 0.72 barriers to participation in physical activity, with 89% of the respondents identifying no barriers to participation.

The barrier that had the highest likelihood of predicting nonparticipation differed between males and females. Males were more likely to be nonparticipants due to the activity not being available in their area, although this needs to be interpreted with some caution given the small sample size. In contrast, women were more likely to be nonparticipants due to time. Time being the most significant barrier to nonparticipation in women is supported by earlier work by Yoshida et al. [21] and Johnson et al. [22]. However, it is in contrast to the findings of O'Neill and Reid [14], where respondents ranked time as the 14th barrier to participation. The participants in O'Neill and Reid also identified "I get tired easily" as the second most frequent barrier. In the current study, a lack of energy was not related to nonparticipation in women.

Self-reported health status was not related to nonparticipation in the current sample. This may not be surprising given that this sample represented only those who did not identify a health condition, illness, or injury as barriers to participation. By limiting the sample to those who perceived their health as not posing a limiting factor, it is difficult to directly compare the results to other studies where health was consistently identified as the number one barrier to participation. What is surprising in the current study, however, is the relationship between chronic health conditions and

Table 2: Barriers by participation (weighted sample).

Variable	Category	*N*	%
Cost	Participant		
	Yes	308	6.9
	No	4130	93.1
	Nonparticipant		
	Yes	26	5.7
	No	433	94.3
Transportation	Participant		
	Yes	147	3.3
	No	4291	96.7
	Nonparticipant		
	Yes	15	3.3
	No	444	96.7
Not available in area	Participant		
	Yes	283	6.4
	No	4156	93.6
	Nonparticipant		
	Yes	14	3.1
	No	445	96.9
Not physically accessible	Participant		
	Yes	50	1.1
	No	4389	98.9
	Nonparticipant		
	Yes	4	0.9
	No	455	99.1
Location is too far	Participant		
	Yes	173	3.9
	No	4266	96.1
	Nonparticipant		
	Yes	8	1.7
	No	451	98.3
Fear of injury	Participant		
	Yes	76	1.7
	No	4363	98.3
	Nonparticipant		
	Yes	5	1.1
	No	454	98.9
Lack of time	Participant		
	Yes	1922	43.3
	No	2517	56.7
	Nonparticipant		
	Yes	184	40.1
	No	275	56.9
Lack of energy	Participant		
	Yes	456	10.3
	No	3982	89.7
	Nonparticipant		
	Yes	72	15.7
	No	387	84.3

Table 2: Continued.

Variable	Category	N	%
Lack of motivation	Participant		
	Yes	1281	28.9
	No	3158	71.1
	Nonparticipant		
	Yes	146	31.8
	No	313	68.2
Lack of skills or knowledge	Participant		
	Yes	34	0.8
	No	4404	99.2
	Nonparticipant		
	Yes	4	0.9
	No	454	99.1

Table 3: Odds of nonparticipation as a function of type of barrier.

Barrier	Males		Females	
	Odds ratios	95% CI	Odds ratios	95% CI
Cost	2.05	0.73, 5.74	1.07	0.67, 1.71
Transportation	0.89	0.16, 5.04	1.03	0.56, 1.88
Not available in area	**4.50***	**1.09, 18.68**	1.50	0.81, 2.78
Location not physically accessible	0.33	0.05, 2.31	1.53	0.39, 5.94
Location is too far	11.96	0.46, 313.09	1.70	0.76, 3.80
Fear of injury	2.29	0.25, 21.50	1.59	0.58, 4.34
Lack of time	0.79	0.56, 1.13	**1.45***	**1.09, 1.92**
Lack of energy	0.91	0.56, 1.48	**0.54***	**0.39, 0.76**
Lack of motivation	**0.69***	**0.48, 0.98**	1.18	0.88, 1.58
Lack of skills/knowledge	0.75	0.07, 8.09	0.78	0.25, 2.40

*$P<0.05$.

Table 4: Odds of nonparticipation as a function of self-rated general health status.

Barrier	Males		Females	
	Odds ratios	95% CI	Odds ratios	95% CI
Excellent/very good	0.74	0.45, 1.20	0.74	0.45, 1.20
Good	0.78	0.46, 1.30	0.79	0.46, 1.30
Fair/poor	1.00	Referent	1.00	Referent

Table 5: Odds of nonparticipation as a function of health condition.

Barrier	Males		Females	
	Odds ratio	95% CI	Odds ratios	95% CI
Vision function	**3.06***	**1.30, 7.21**	0.25	0.37, 1.67
Heart disease	1.03	0.75, 1.41	**1.54***	**1.19, 1.99**
COPD	0.76	0.33, 1.77	**2.11***	**1.34, 3.32**
Diabetes	**1.66***	**1.12, 2.44**	**1.85***	**1.30, 2.63**
Osteoporosis	0.42	0.11, 1.63	0.94	0.69, 1.28
Effects of a stroke	1.07	0.36, 3.21	1.42	0.51, 3.98
Mobility trouble	**2.65***	**1.01, 6.95**	**2.71***	**1.70, 4.31**

*$P < 0.05$.

nonparticipation. These results suggest that while Canadian seniors have underlying chronic health conditions they do not always identify them as being associated with their self-reported general health, nor do they view them as limiting conditions to participation in physical activity. This potential disconnect between self-reported health status and chronic health conditions is interesting, as heart disease, vision, COPD, diabetes, and mobility trouble increased the likelihood of nonparticipation across males and females. It is possible that these older adults have learned to compensate for these chronic health conditions in their everyday life and therefore no longer consider them barriers to participation nor consider them impacting their self-reported general health status. It is clear, however, that there are limiting factors to participation. Although health concerns have been considered motivators to participation [23], this does not seem to be the case in the current study.

These findings have implications for physical activity programming for seniors. While only a small number of older Canadians were identified who did not participate, this sample represented those who believed they did not have limitations to participation; effectively creating a "healthy" sample of older adults. Regardless of their self-perceptions, this group of seniors was less likely to participate because of underlying health conditions. This would suggest that programming should target the specific needs of older adults; whether that takes the form of activities and classes designed specifically for those with targeted health conditions, or increased knowledge of how to integrate and attract those with health conditions into preexisting classes. What is clear is that the results identify a dissociation between self-perceptions of health and the reasons older adults are at greater risk for nonparticipation.

This study is not without limitations. The derived variable we chose from the CCHS-HA dataset captures physical activity as any one of walking for pleasure or exercise, light sports, moderate sports, strenuous sports, and exercises to increase muscle strength and endurance. While this variable is all encompassing for physical activity, it does not include activities of daily living, which may also be considered by some to contribute to their daily physical activity levels. Second, the self-reporting of general health status does not take into account the fact that many older adults learn to accommodate health concerns and to compensate for their impact on daily activities. Thus, a physical performance based measurement of health status could quite possibly have produced a different pattern of findings. Third, the chronic health conditions selected were done so to represent an overall picture of health-related issues that may impact participation. Specifically, they were chosen to represent vision, respiratory function, cardiovascular health, neurological health, diabetes, and mobility. It is by no means an exhaustive list of all chronic health conditions; any of which could impact ones' ability to participate in physical activity. The chronic health conditions were not validated in any way through performance measures, suggesting that their presence may have been under- or overrepresented in the self-reporting. Fourth, in predicting nonparticipation, the overall sample of nonparticipants represented only 9.4% of the total sample derived. Thus, the potential for type I error may be increased. Lastly, it was not possible to determine whether participants were meeting the recommended guidelines for participation in physical activity. While the PASE scores confirmed this group was more active than the nonparticipants, it was not possible to determine if this was due to frequency of participation or the duration of participation.

Overall this paper raises the issue that despite having created a "healthy" sample of older Canadians, there are still those who do not participate in regular physical activity. The 9.4% of seniors in this sample who did not participate is substantially smaller than the national average of 62%. However, it is important to note that this sample has been derived from older adults who reported no health condition or limitation preventing them from participating and thus is not representative of the general population of older adults. As such, their nonparticipation is not related to the presence of barriers such as opportunity or desire, but to specific chronic health conditions, and suggests a potential disconnect between self-perceived health and actual health status. Considering that only one-third or less of older Canadians achieve the recommended guidelines for leisure time physical activity [4], it is important to shift the focus from simply describing the barriers and motivators to physical activity to working towards designing programs that will address the health conditions facing older Canadians. With the changing age demographic, it will be important to encourage all older adults to participate in some form of physical activity on a daily basis, including a renewed focus on the role that activities of daily living can play in achieving fitness goals. One positive note to take from our findings is that 90% of the healthy older adults identified do engage in some form of physical activity, suggesting that public health messaging is reaching its mark.

Acknowledgment

This research was funded by the Social Sciences and Humanities Research Council of Canada, Grant no. 862-2010-0007 (P. L. Weir).

References

[1] Statistics Canada, "Population Projections for Canada, Provinces and Territories," http://www.statcan.gc.ca/pub/91-520-x/2010001/aftertoc-aprestdm1-eng.htm, 2010.

[2] V. A. Hughes, W. R. Frontera, M. Wood et al., "Longitudinal muscle strength changes in older adults: influence of muscle mass, physical activity, and health," *Journals of Gerontology*, vol. 56, no. 5, pp. B209–B217, 2001.

[3] W. J. Chodzko-Zajko, D. N. Proctor, M. A. Fiatarone Singh et al., "Exercise and physical activity for older adults," *Medicine and Science in Sports and Exercise*, vol. 41, no. 7, pp. 1510–1530, 2009.

[4] M. C. Ashe, W. C. Miller, J. J. Eng, and L. Noreau, "Older adults, chronic disease and leisure-time physical activity," *Gerontology*, vol. 55, no. 1, pp. 64–72, 2009.

[5] Seniors in Canada Report Card, National Advisory Council on Aging, Public Health Agency of Canada, Division of Aging and Seniors, 2006.

[6] A. E. Mathews, S. B. Laditka, J. N. Laditka et al., "Older adults' perceived physical activity enablers and barriers: a multicultural perspective," *Journal of Aging and Physical Activity*, vol. 18, no. 2, pp. 119–140, 2010.

[7] K. A. Schutzer and B. S. Graves, "Barriers and motivations to exercise in older adults," *Preventive Medicine*, vol. 39, no. 5, pp. 1056–1061, 2004.

[8] J. Cohen-Mansfield, M. S. Marx, and J. M. Guralnik, "Motivators and barriers to exercise in an older community-dwelling population," *Journal of Aging and Physical Activity*, vol. 11, no. 2, pp. 242–253, 2003.

[9] F. D. Lees, P. G. Clark, C. R. Nigg, and P. Newman, "Barriers to exercise behavior among older adults: a focus-group study," *Journal of Aging and Physical Activity*, vol. 13, no. 1, pp. 23–33, 2005.

[10] S. O'Brien Cousins, "'My heart couldn't take it': older women's beliefs about exercise benefits and risks," *Journals of Gerontology B*, vol. 55, no. 5, pp. P283–P294, 2000.

[11] M. P. Buman, L. Daphna Yasova, and P. R. Giacobbi, "Descriptive and narrative reports of barriers and motivators to physical activity in sedentary older adults," *Psychology of Sport and Exercise*, vol. 11, no. 3, pp. 223–230, 2010.

[12] M. Rasinaho, M. Hirvensalo, R. Leinonen, T. Lintunen, and T. Rantanen, "Motives for and barriers to physical activity among older adults with mobility limitations," *Journal of Aging and Physical Activity*, vol. 15, no. 1, pp. 90–102, 2007.

[13] R. N. Butler, R. Davis, C. B. Lewis, M. E. Nelson, and E. Strauss, "Physical fitness: exercise prescription for older adults," *Geriatrics*, vol. 53, no. 11, pp. 45–56, 1998.

[14] K. O'Neill and G. Reid, "Perceived barriers to physical activity by older adults," *Canadian Journal of Public Health*, vol. 82, no. 6, pp. 392–396, 1991.

[15] C. M. Connell, R. M. Davies, A. M. Rosenberg, and E. B. Fisher Jr., "Retiree's perceived incentives and barriers to participation in health promotion activities," *Health Education Research*, vol. 3, no. 3, pp. 325–330, 1988.

[16] M. Denton, S. Prus, and V. Walters, "Gender differences in health: a Canadian study of the psychosocial, structural and behavioural determinants of health," *Social Science and Medicine*, vol. 58, no. 12, pp. 2585–2600, 2004.

[17] P. McDonough and V. Walters, "Gender and health: reassessing patterns and explanations," *Social Science and Medicine*, vol. 52, no. 4, pp. 547–559, 2001.

[18] F. Orfila, M. Ferrer, R. Lamarca, C. Tebe, A. Domingo-Salvany, and J. Alonso, "Gender differences in health-related quality of life among the elderly: the role of objective functional capacity and chronic conditions," *Social Science and Medicine*, vol. 63, no. 9, pp. 2367–2380, 2006.

[19] Statistics Canada, Canadian Community Health Survey (CCHS-HA), Healthy Aging. User Guide, 1-65, 2010.

[20] R. A. Washburn, E. McAuley, J. Katula, S. L. Mihalko, and R. A. Boileau, "The physical activity scale for the elderly (PASE): evidence for validity," *Journal of Clinical Epidemiology*, vol. 52, no. 7, pp. 643–651, 1999.

[21] K. K. Yoshida, K. R. Allison, and R. W. Osborn, "Social factors influencing perceived barriers to physical exercise among women," *Canadian Journal of Public Health*, vol. 79, no. 2, pp. 104–108, 1988.

[22] C. A. Johnson, S. A. Corrigan, P. M. Dubbert, and S. E. Gramling, "Perceived barriers to exercise and weight control practices in community women," *Women and Health*, vol. 16, no. 3-4, pp. 177–191, 1990.

[23] R. S. Newson and E. B. Kemps, "Factors that promote and prevent exercise engagement in older adults," *Journal of Aging and Health*, vol. 19, no. 3, pp. 470–481, 2007.

Hindawi Publishing Corporation
Journal of Aging Research
Volume 2012, Article ID 468156, 8 pages
doi:10.1155/2012/468156

Research Article

Physical Activity across Frailty Phenotypes in Females with Parkinson's Disease

Kaitlyn P. Roland,[1] Kayla M. D. Cornett,[1] Olga Theou,[2] Jennifer M. Jakobi,[3] and Gareth R. Jones[3]

[1] *Interdisciplinary Graduate Studies, Health and Exercise Sciences, University of British Columbia Okanagan, Kelowna, BC, Canada V1V 1V7*
[2] *Faculty of Geriatrics Medicine, Dalhousie University, Halifax, NS, Canada B3H 2E1*
[3] *School of Health and Exercise Sciences, Faculty of Health and Social Development, University of British Columbia Okanagan, 3333 University Way, Kelowna, BC, Canada V1V 1V7*

Correspondence should be addressed to Gareth R. Jones, gareth.jones@ubc.ca

Received 3 February 2012; Revised 2 June 2012; Accepted 21 June 2012

Academic Editor: Barbara Tettenborn

Females with Parkinson's disease (PD) are vulnerable to frailty. PD eventually leads to decreased physical activity, an indicator of frailty. We speculate PD results in frailty through reduced physical activity. *Objective*. Determine the contribution of physical activity on frailty in PD ($n = 15$, 65 ± 9 years) and non-PD ($n = 15$, 73 ± 14 years) females. *Methods*. Frailty phenotype (nonfrail/prefrail/frail) was categorized and 8 hours of physical activity was measured using accelerometer, global positioning system, and self-report. Two-way ANCOVA (age as covariate) was used to compare physical activity between disease and frailty phenotypes. Spearman correlation assessed relationships, and linear regression determined associations with frailty. *Results*. Nonfrail recorded more physical activity (intensity, counts, self-report) compared with frail. Self-reported physical activity was greater in PD than non-PD. In non-PD, step counts, light physical activity time, sedentary time, and self-reported physical activity were related to frailty ($R = 0.91$). In PD, only carbidopa-levodopa dose was related to frailty ($r = 0.61$). *Conclusion*. Physical activity influences frailty in females without PD. In PD females, disease management may be a better indicator of frailty than physical activity. Further investigation into how PD associated factors contribute to frailty is warranted.

1. Introduction

Frailty is a geriatric syndrome that results in an increased vulnerability to acute and chronic illness, falls and related injuries, and a general loss of functional independence [1–4]. The cardiovascular health study frailty index (CHS*fi*), proposes that frailty develops across a spectrum of phenotypes ranging from nonfrail to prefrail to frail [5]. All stages of frailty are evident within community-dwelling populations, with over 70% of older adults expressing some frailty characteristics [6]. Frailty is twice as prevalent in females as males and females typically display more frailty characteristics than males [5, 7]. Females experience increased frailty severity than males since they live longer and spend a greater proportion of life managing disability and disease [7–9].

Females with Parkinson's disease (PD) are at an increased risk of frailty [10], yet the presence of frailty in persons with PD may be misinterpreted as disease-related functional decline [11]. Females with PD are typically older [12], cite greater disability [13] and experience more difficulty performing activities of daily living (ADL) than males with PD [14, 15]. Recent evidence suggests that of the five CHS*fi* criteria, self-reported exhaustion best determines frailty phenotype in females with PD [10]. Exhaustion is a common complaint among older females, especially those with PD, and is strongly associated with inhibiting physical activity and ADL participation [16–18]. The established relationship between reduced physical activity and frailty severity [19] leads us to speculate that PD influences frailty through decreased participation in physical activity. Understanding how physical activity contributes to frailty in females with PD is important in directing management strategies aimed at maintaining functional independence. The objective of this study was to determine the contribution of daily physical activity on frailty phenotype in community-dwelling females with PD, compared to non-PD females.

2. Methods

Females greater than 50 years of age, living independently in the community, with mild to moderate PD severity (Hoehn & Yahr stage 1–3) were recruited through advertisements and support group presentations. Non-PD females were similarly recruited from the same local region. All participants were able to ambulate independently (with or without walk-aid). Females with PD were in a steady clinical state (controlled by medication) and cognitively intact. Females with PD were instructed to continue daily dopamine medication as prescribed and no incidence of freezing of gait or severe dyskinesia episodes were reported during the testing day. All participants provided written informed consent. The Clinical Research Ethics Board of the University of British Columbia granted ethical approval for this investigation.

Health history questionnaires, physical frailty criteria (CHS*fi*), self-reported physical activity, and setup of daily physical activity monitors (accelerometer, GPS) were completed at the participant's home in the morning (8~10 am). All PD participants were assessed between 1 and 2 hours post anti-Parkinson's medication, and controls were assessed 1 hour after breakfast. In older adults, reduction in daily physical activity, and associated physiological change, can be quantified using continuous objective physical activity monitors (i.e., accelerometers and global positioning systems, GPS) [20]. In this study, a waist-borne accelerometer and wrist-born GPS recorded physical activity; and the participant wore these devices continuously for the entire testing day. The participant was then instructed to go about their typical daily activities, which they recorded in a written hourly log. The accelerometer, GPS, and physical activity logbook were collected approximately 7 hours later (between 4–7 pm) at the participant's home.

2.1. Frailty Phenotype. Frailty was categorized according to CHS*fi* [5] that includes five select criteria to determine a frailty phenotype (nonfrail, prefrail, and frail). These criteria include: (1) Unintended weight loss (>10 lbs in past 12 months); (2) Weakness (maximal handgrip strength classified by body mass index, BMI); (3) Walk speed (15 ft at usual pace classified by height); (4) Self-reported energy expenditure (Minnesota Leisure Time Activities Questionnaire, MLTA); (5) Self-reported exhaustion (Center for Epidemiological Studies depression scale, CES-D). Participants were considered nonfrail if they satisfied none of the phenotypic criteria, prefrail if they satisfied 1 or 2 criteria, and frail if they satisfied 3 or more criteria [5].

2.2. Accelerometer. Daily physical activity was measured using an ActiTrainer accelerometer (Actigraph, LLC, Fort Walton Beach, FL) secured in a holster worn at the waist on the dominant side. The ActiTrainer ($8.6 \times 3.3 \times 1.5$ cm; 51 grams) is a triaxial solid-state accelerometer that was programmed to record 60-second epochs of data. Data was uploaded to ActiLife5 v.5.8.3 software (ActiGraph, LLC, Fort Walton Beach, FL). Physical activity intensity levels were categorized according to the cut-points described by Copeland and Eslinger for older adults [21]. Sedentary activity was defined as 0 to 50 counts per minute, light physical activity as 51 to 1,040 counts per minute, and moderate-to-vigorous physical activity as greater than 1,041 counts per minute. Percentage of time spent at each level of activity was reported. Measurement outputs included total counts (i.e., daily step and activity counts, total minutes of activity) and intensity (i.e., percentage of time spent at sedentary, light, and moderate-vigorous activity intensity levels).

2.3. Global Positioning System (GPS). Global Positioning System (GPS) examines gross mobility outside the individuals' home. GPS used in combination with the accelerometer can accurately assess physical activity within real-life environments [22], and this can be applied to categorize stages of frailty [23]. Participants wore a Garmin Forerunner 405 GPS watch (Garmin International Inc., Olathe, KS). GPS data were uploaded to the Garmin training center software (http://connect.garmin.com/). Both the GPS and accelerometer units were synchronized to record minute-by-minute data. The GPS and accelerometer data were time-matched using the ActiGraph GPS Correlation Wizard v.1.0.0 (Actigraph, LLC, Fort Walton Beach, FL) and exported in a Microsoft Excel compatible format for analysis. Only physical activity completed outside the home, defined using Garmin Training Software and an accompanying Google Earth Map, was included in the analysis. Participants' hourly physical activity log was also compared to GPS outputs to confirm physical activity participation. Vehicle-generated activity was considered any recording that measured greater than 3 m/sec for >1 min. All activity at speeds less than 3 m/sec >1 min were included as participant physical activity. GPS measurement outputs included; total GPS distance (km, vehicle- and participant-generated) and total amount of physical activity time (min).

2.4. Self-Reported Physical Activities. Self perceived energy expenditure was assessed using the self-reported MLTA [24], which was also used to determine energy expenditure as part of the CHS*fi* frailty assessment [5]. Twenty activities were specified, including walking for exercise, moderately strenuous household chores, mowing the lawn, raking the lawn, gardening, hiking, jogging, biking, exercise cycling, dancing, aerobics, bowling, golfing, calisthenics/general exercise, swimming, doubles tennis, singles tennis, and racquetball. Participants who engaged in any physical activity during the previous 2-weeks recorded the number of sessions and their duration. Energy (kcal/week) expenditure was determined using metabolic equivalent (MET) score: (activity-specific MET) × ((activity duration in minutes)/60) × ((number of sessions in past two weeks)/2). Total energy expenditure was calculated by summing expenditures over all activities.

2.5. Statistical Analysis. All analysis was performed using Statistical Package for Social Science 18.0 (PASW Statistics 18.0, SPSS Inc. IBM Somers, NY). Subject characteristics were compared between groups (non-PD, PD) and between frailty phenotype (nonfrail, prefrail, and frail) with a one-way ANOVA. The non-PD females were older than PD ($P = 0.005$), nonfrail females were younger than prefrail and frail ($P = 0.007$), and prefrail were younger than frail ($P = 0.03$). Thus, age was used as a covariate in a two-way ANCOVA to assess differences in physical activity between disease states (PD; non-PD) and frailty phenotype (nonfrail; prefrail; frail). To evaluate the main effects a univariate analysis was performed for each dependent variable to identify contributions to the main effects of disease states and frailty phenotypes. Probability level was set at $P < 0.05$ and Tukey post hoc tests were used to probe statistical interactions. Spearman's rank correlation was used to evaluate physical activity measures relative to frailty phenotype in each group. The physical activity measures that were significantly correlated with frailty phenotype were entered into a multiple regression analysis model with frailty phenotype as the dependent variable and physical activity measures as the independent variables.

3. Results

Fifteen persons with PD (65 ± 9 years) and 15 non-PD controls (73 ± 14 years) participated. Both groups were categorized into frailty phenotypes according to the CHS*fi* (Table 1). In females with PD, tremor in the upper limb was controlled with medication and participants self-reported no freezing of gait or periods of dyskinesia over the course of the day. Further, any reports of rigidity and/or slowness of movement (bradykinesia) were mild and did not restrict ADL. Comparison of physical activity between disease states and frailty phenotypes with age as a covariate determined nonfrail recorded less sedentary time, participated in more light-intensity physical activity and accumulated more steps compared with frail (Table 2). Also, self-reported physical activity was lower in frail compared with the nonfrail and prefrail phenotypes (Table 2). However, higher self-reported physical activity was reported in PD compared with non-PD, although the other physical activity variables did not differ between disease groups (Table 2).

3.1. Physical Activity and Frailty. In PD, no physical activity variables were significantly related to frailty ($P > 0.29$); however, daily dose of carbidopa-levodopa, including both controlled-release and active-release forms, was correlated with frailty ($r = 0.61$; $P = 0.01$) (Figure 1). Other medication regimes for the management of PD and other comorbidities did not relate to frailty ($P > 0.42$). Physical activity variables demonstrated a significant linear relationship with increasing frailty severity in non-PD (Table 3). Those physical activity variables that were significantly different between frailty phenotypes (Table 3) were entered into individual regression models for non-PD. Low step, counts, higher sedentary behaviour, reduced light activity and lower weekly self-reported energy expenditure accounted for 83.3% of the variance ($R = 0.913$; $P = 0.002$; Figures 2(a)–2(d), but no single variable in this model best-determined frailty ($P = 0.108$ to 0.226).

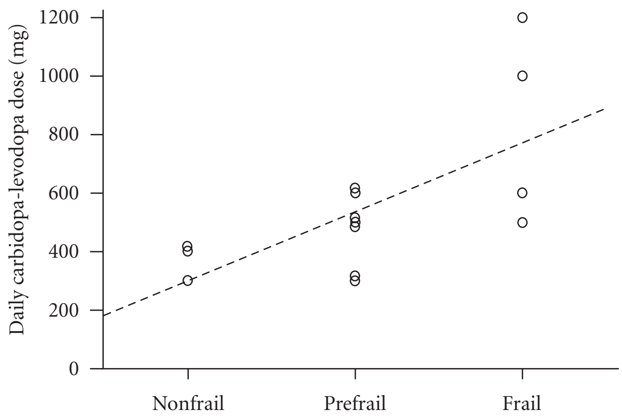


Figure 1: Relationship between frailty severity, measured by Cardiovascular Health Study frailty index, and daily carbidopa-levodopa dose (mg). Increased step count is positively correlated with greater frailty severity ($r = 0.61$) in females with PD. Females with PD are represented by the *open circles* and *dashed regression line*.

4. Discussion

This study examined the relationship between physical activity and frailty phenotype in community-dwelling females with PD and non-PD controls. Results of this study demonstrate physical activity (i.e., accelerometry counts and intensity, GPS, and self-reported) was not related to frailty phenotype in this sample group of females with PD; however, lower daily physical activity was associated with increased frailty severity in non-PD females. Current literature demonstrates physical inactivity as an important determinant of frailty phenotype [25], and persons with PD have reduced levels of accelerometry-assessed physical activity compared to non-PD controls [26]. This study suggests daily physical activity participation may not be the primary factor associated with frailty phenotype in females with PD. It is unclear from our cross-sectional data if a causal relationship exists between physical activity and frailty. However, results presented here support future longitudinal investigation into how PD progression impacts physical activity and how these changes in symptom expression and physical activity influence physical frailty.

4.1. Physical Activity: Not Related to Frailty Phenotype in PD. No relationship was demonstrated between daily physical activity and frailty phenotype in these females with PD. Although PD progression eventually debilitates motor performance, it is not likely to be the primary contributing factor to frailty in these females with PD. The results presented here do not necessarily exclude the contribution of physical activity to frailty, but rather suggest disease management may be a greater contributor to frailty. There is considerable variation in the manifestation of PD symptoms,

Table 1: Participant characteristics across frailty phenotypes in PD and non-PD females.

	PD			Non-PD		
	Nonfrail	Prefrail	Frail	Nonfrail	Prefrail	Frail
N	4	7	4	8	4	3
Age	69 ± 1	65 ± 10	63 ± 11	63 ± 8[ab]	79 ± 14[b]	90 ± 6
Body mass index (kg/m^2)	26.31 ± 5.6	22.95 ± 4.3	25.06 ± 4.3	23.20 ± 5.7	34.60 ± 5.4	32.49 ± 12.2
Number of medications	4.0 ± 2.0	2.9 ± 1.5	3.3 ± 2.6	1.3 ± 1.3	6.0 ± 4.2	5.3 ± 0.6
mg carbidopa-levodopa per day	366.67 ± 57.7	571.43 ± 340.2	825.00 ± 330.4			
carbidopa-levodopa only (N)	2	3	2			
carbidopa-levodopa + pram (N)	2	2	0			
carbidopa-levodopa + enta (N)	0	0	1			
carbidopa-levodopa + enta + pram (N)	0	1	1			
carbidopa-levodopa + aman + rop (N)	0	1	0			
Hoehn & Yahr disease severity	1.83 ± 0.8	1.86 ± 0.6	2.50 ± 0.4			

PD: Parkinson's disease, N: number, kg: kilogram, m: meter, mg: milligram, pram: pramipexole, enta: entacapone, aman, amantadine, and rop: ropinirole.
[a]Significantly different from prefrail.
[b]Significantly different from frail.

Table 2: Main effects on physical activity variables.

	PD	Non-PD	Nonfrail	Prefrail	Frail
Number	15	15	12	11	7
Total steps	3476 ± 2814	3731 ± 3827	5624 ± 3309[a]	3019 ± 3290	1636 ± 1599
% time spent sedentary	61.7 ± 14.1	60.9 ± 16.6	49.39 ± 9.5[a]	66.4 ± 14.6	71.9 ± 11.3
% time at light activity	32.2 ± 10.6	31.7 ± 10.6	39.4 ± 7.4[a]	28.6 ± 10.2	25.2 ± 8.2
MLTA questionnaire	3052.3 ± 1611.6[b]	2015.0 ± 1517.4	3045.0 ± 1096.2[a]	2826.4 ± 1790.4[a]	1196.1 ± 1493.7

Analysis adjusted for age.
%: percent, MLTA: Minnesota Leisure Time Activity, and PD: Parkinson's disease.
[a]Significantly different from frail, $P < 0.05$.
[b]Significantly different from non-PD, $P = 0.03$.

Table 3: The relationship of physical activity variables to frailty severity in non-PD females.

	ANOVA	Spearman correlation		Linear regression	
	Main effect (P)	Correlation coefficient (r)	Significance (P)	Beta coefficient (β)	Significance (P)
Accelerometer: total counts					
Total steps counts*	0.046	−0.79	0.001	1.113	0.202
Total activity counts	NS	−0.75	0.001		
Total activity time (min)	NS	−0.59	0.001		
Accelerometer: intensity					
% time spent sedentary*	0.002	0.84	0.000	2.602	0.110
% time at light activity*	0.012	−0.82	0.000	1.225	0.226
% time at MV activity	NS	−0.81	0.000		
GPS					
Total distance travelled (km)	NS	−0.63	0.012		
Average travel speed (km/h)	NS	−0.58	0.023		
Physical activity time (min)	NS	−0.54	0.036		
Self-reported activity					
MLTA questionnaire (kcal/week)*	0.007	−0.82	0.000	−0.386	0.108

%: percentage; GPS: global positioning system; h: hour; kcal: kilocalories; km: kilometers; MLTA: Minnesota Leisure Time Activity; min: minutes; MV: moderate-vigorous; PD: Parkinson's disease; NS: nonsignificant ($P > 0.05$).
*Significant main effects, therefore included in regression model.

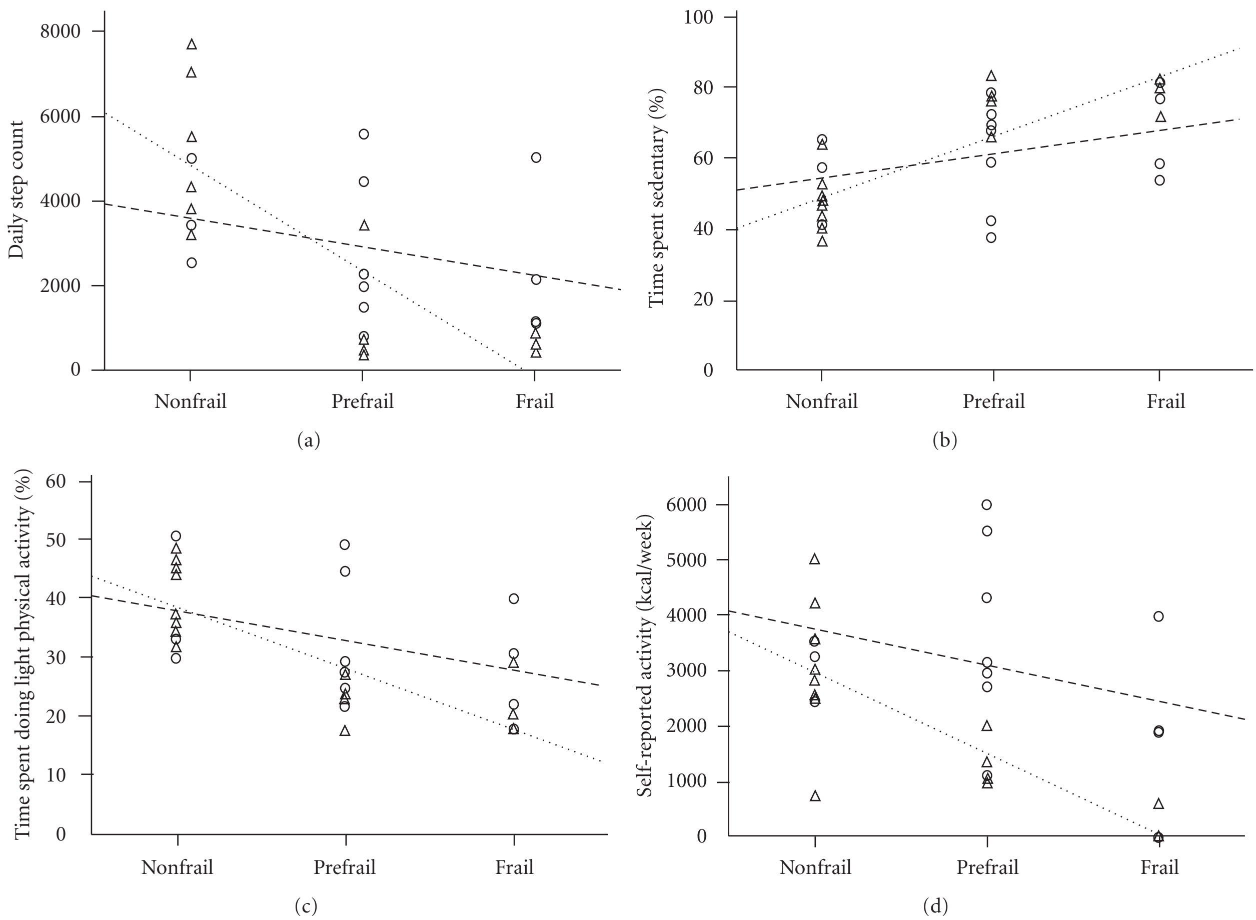


Figure 2: Relationship between frailty severity and: (a) daily step count. Total daily step count was negatively correlated with frailty severity ($r = -0.79$) in females without PD; (b) accumulated daily sedentary time. Sedentary time was positively correlated with frailty severity ($r = 0.84$) in females without PD; (c) accumulated light physical activity time. Light activity time was negatively correlated with frailty severity ($r = -0.82$) in females without PD; (d) self-reported leisure activity. Self-reported activity was negatively correlated with greater frailty severity ($r = -0.82$) in females without PD. Females with PD are represented by the *open circle* and *dashed regression line*; females without PD are represented by *the open triangle* and *dotted regression line*.

which necessitates further large-scale investigation into PD-related contributors to frailty over the disease course. During the first 10 years of the disease, symptoms such as akinesia and festinating gait may not have progressed to the point at which they inhibit physical activity participation, which was the case with these participants [27]. In the initial stages of the disease, slowness to execute day-to-day activities (i.e., bradykinesia) is more common, and this likely contributes to perceived exhaustion, which is a criterion for frailty. Thus, in community-dwelling persons with PD, self-reported exhaustion resulting from PD symptoms contributes to frailty [10], rather than reduced daily physical activity.

Results of this study indicate that although physical activity was not related to frailty in PD, females with PD self-reported greater leisure time activities compared to non-PD controls, regardless of frailty severity. This finding argues against previous research that states community-dwelling persons with PD have significantly less daily physical activity energy expenditure (measured using MLTA questionnaire) compared with controls, although the study sample included only males [28]. We speculate that increased self-reported leisure activity in these females with PD is due to the benefits of physical exercise being well recognized in the PD population to improve motor performance, functional and cognitive ability, safety, and confidence in ADL [27]. Persons living with PD are constantly encouraged to remain active despite disease-associated barriers (i.e., exhaustion) [29, 30]. Previous study demonstrated older adults with an increased risk of mortality, like persons with PD, adhere better to exercise programs compared to general community-dwelling older adults [31]. Therefore, we speculate that persons with PD may be involved in more physical activity and adhere better to exercise recommendations compared with community-dwelling counterparts because of PD-associated disease symptoms, regardless of frailty phenotype.

Unlike females with PD, physical activity influences frailty severity in community-dwelling females without PD. The importance of dedicating a greater percentage of daytime hours to light-intensity activities and decreasing sedentary time is highlighted in community-dwelling females.

Sedentary time was related to increased frailty in females without PD, likely through its influence on physiological decline (i.e., decreased fitness), leading to greater dependence [32]. The contribution of physical activity to frailty in females without PD may be attributed to reduced mobility, less activity intensity and restricted life-space (i.e., the spatial area a person moves through during daily life) [33, 34]. These physical and environmental challenges contribute to further reductions in physical activity and exacerbate declining functional reserves [35], encouraging transition to greater frailty severity. Remaining active during old age is important as sedentary lifestyle significantly increases risk of developing multiple chronic diseases and premature mortality [35]. Disease prevalence and functional impairment increases with age [36], which places the older adult at greater risk of frailty [5]. However, it is unclear whether age and age-related characteristics are associated with frailty in females with PD. This sample was randomly recruited from a population of females living independently in their own homes. Persons with PD seek institutional care sooner than non-PD counterparts [37], suggesting they may reach frailty sooner. Future longitudinal research may investigate onset of frailty in females with PD and follow frailty progression throughout the disease course. Also, further information on the contribution of physical activity to frailty onset and progression in PD is needed.

4.2. The Relationship between PD Management and Frailty. Accelerometers have been used in several research fields to monitor daily physical activity; monitoring of steps using waist-born accelerometers is feasible in PD populations and provides useful feedback on freezing (i.e., sudden inability to move, especially in the legs during walking), as well as long-term daily activity. Results of this study indicate that daily physical activity does not contribute to frailty phenotype in community-dwelling females with PD; however, daily carbidopa-levodopa dose was significantly related to frailty. Medication regimes for participants in this study included medications for the management of PD (i.e., carbidopa-levodopa, entacapone, pramipexole, amantadine, and ropinerole) and other conditions, such as anxiety/depression, blood pressure, migraine, muscle pain and inflammation, postmenopause symptoms, and difficulty sleeping. Multiple medication use in PD is associated with functional decline and high fall risk [38]; both of which are indicative of frailty. In addition, dopamine deficiency in PD may result in physical exhaustion [39], increasing ADL dependence risk for frailty. Therefore, the impact of PD medication regime on physical function and frailty requires further inquiry.

In addition to motor impairments, persons with PD face secondary symptoms that impact basic daily function, such as depression, cognitive impairments, and nonmotor symptoms that increase functional dependence through increased anxiety, social isolation, and confusion [40]. Cognition is considered an important component of frailty [41] and is also associated with adverse PD outcomes [40]. Cognitive function can be influenced by depression and physical decline, and females with PD report greater incidence of both compared with males [14]. Also, females with PD report greater symptom-related stress and sleep disturbances than males [42, 43]. The importance of these non-motor symptoms cannot be underestimated, however they are beyond the scope of this study. Future research should consider how PD symptoms (motor and non-motor), disease severity, duration, other comorbidities, polypharmacy, and age impact frailty severity in both males and females with PD.

4.3. Implications

4.3.1. Community-Dwelling Populations and Neurological Disorders. The progressive nature of PD and related symptoms such as bradykinesia and tremor causes persons with PD to seek long-term care earlier than the general older adult population [37]. Females with PD may be more vulnerable to frailty than persons without neurological disorder because of PD-related systems that exacerbate the frailty phenotype. Therefore, it is important to make an early identification of frailty in community dwelling persons, especially those with neurological disorder who express a frailty phenotype earlier than the general population.

4.3.2. Frailty Management. Physical inactivity is directly linked to declining physiological reserve capacity, defined as adaptive responses (i.e., heart rate, blood pressure, respiration) that enable us to perform tasks or overcome external stresses [44]. In persons with PD the complexity of symptom presentation results in a reduced adaptive capacity. These losses of adaptive capacity, related to aging or disease, lead to declining functional independence and are a determinant of frailty [25]. Comorbidities and clinical symptoms interact between frailty and PD making identification of frailty in persons with PD challenging to diagnose [11]. Few studies have examined how to accurately identify frailty in this population [45, 46]. Early identification of frailty in persons with PD is relevant since over half of older adults living independently in their own homes are at-risk for frailty and subsequent functional decline [6]. Due to the transitional nature of both frailty and PD, the majority of disease progression occurs long before the individual requires institutional care [47]. As PD severity increases, symptoms become aggravated, further exacerbating underlying frailty [46]. Awareness of frailty during the initial stages of PD development may contribute to improved management strategies that delay and/or reverse frailty factors and preserve functional independence [6]. This study demonstrated that frailty was related to decreased physical activity in community-dwelling non-PD, and symptom management in PD females. Knowledge gained from the current study can be used to inform effective strategies for identifying prefrailty in non-PD females. This information can be applied to the development and delivery of timely support that addresses age- and disease-associated declines in function. Preventing functional decline has important implications for healthcare resource use in PD and non-PD older adults, as well as reducing physical, emotional, social, and financial problems attributed to frailty [25].

Currently, there is little evidence on how PD specifically impacts females and contributes to increased risk of frailty. Greater risk of frailty may be a consequence of greater functional declines [10], distress, and cognitive impairment [41], all resulting in ADL dependence [8]. In females with PD, physical activity participation should be aimed at managing PD symptoms. Managing PD symptoms, through physical activity and medication, will incidentally contribute to frailty management. Emphasis, therefore, should be placed on managing frailty within the context of PD symptoms [11]. Further collaboration is crucial between neurological, geriatric practice, and physical therapy/rehabilitation in terms of frailty assessment, progression, and addressing complications resulting in declining physical activity that may culminate in frailty.

5. Conclusion

Physical activity influences frailty expression in older females without PD, surprisingly no relationship between physical activity and frailty was found in our sample of females with PD. In PD, disease management may better indicate frailty severity. Further study is warranted to establish how PD-associated characteristics (i.e., polypharmacy) contribute to frailty and how physical activity participation interplays with the complex progress of frailty within PD. This study suggests PD-associated symptoms motivate community-dwelling females with PD to engage in leisure-time physical activities. Considering its vast impact in the community and on healthcare resources, identification of early frailty and management of resulting disability remains a priority area for geriatric research. Ultimately, enabling older adults to remain physically active promotes independence in ADL and empowers positive aging, albeit it may not protect females with PD from becoming frail.

Acknowledgments

The authors would like to thank the participants for generously donating their time and contributing to this investigation. K. P. Roland is funded by the Canadian Institute of Health Research (CIHR) and Parkinson Society Canada's Psychosocial Doctoral Research Award.

References

[1] H. Bergman, F. Béland, S. Karunananthan, S. Hummel, D. Hogan, and C. Wolfson, "Development of a framework for understanding and studying frailty," *Gerontologie et Societe*, vol. 109, no. 2, pp. 15–29, 2004.

[2] L. L. Borges and R. L. Menezes, "Definitions and markers of frailty: a systematic review of literature," *Reviews in Clinical Gerontology*, vol. 21, no. 1, pp. 67–77, 2011.

[3] M. J. Levers, C. A. Estabrooks, and J. C. Ross Kerr, "Factors contributing to frailty: literature review," *Journal of Advanced Nursing*, vol. 56, no. 3, pp. 282–291, 2006.

[4] H. E. Whitson, J. L. Purser, and H. J. Cohen, "Frailty thy name is... Phrailty?" *Journals of Gerontology*, vol. 62, no. 7, pp. 728–730, 2007.

[5] L. P. Fried, C. M. Tangen, J. Walston et al., "Frailty in older adults: evidence for a phenotype," *Journals of Gerontology*, vol. 56, no. 3, pp. M146–M156, 2001.

[6] T. M. Gill, E. A. Gahbauer, H. G. Allore, and L. Han, "Transitions between frailty states among community-living older persons," *Archives of Internal Medicine*, vol. 166, no. 4, pp. 418–423, 2006.

[7] M. T. E. Puts, P. Lips, and D. J. H. Deeg, "Sex differences in the risk of frailty for mortality independent of disability and chronic diseases," *Journal of the American Geriatrics Society*, vol. 53, no. 1, pp. 40–47, 2005.

[8] S. G. Leveille, B. W. J. H. Penninx, D. Melzer, G. Izmirlian, and J. M. Guralnik, "Sex differences in the prevalence of mobility disability in old age: the dynamics of incidence, recovery, and mortality," *Journals of Gerontology*, vol. 55, no. 1, pp. S41–S50, 2000.

[9] A. B. Mitnitski, X. Song, and K. Rockwood, "The estimation of relative fitness and frailty in community-dwelling older adults using self-report data," *Journals of Gerontology*, vol. 59, no. 6, pp. M627–M632, 2004.

[10] K. P. Roland, J. M. Jakobi, C. Powell, and G. R. Jones, "Quality of life as a determinant of frailty phenotype in persons with Parkinson's disease," *Journal of the American Geriatrics Society*, vol. 60, no. 3, pp. 590–592, 2012.

[11] C. Powell, "Frailty and Parkinson's disease: theories and clinical implications," *Parkinsonism and Related Disorders*, vol. 14, no. 4, pp. 271–272, 2008.

[12] D. Twelves, K. S. M. Perkins, and C. Counsell, "Systematic review of incidence studies of Parkinson's disease," *Movement Disorders*, vol. 18, no. 1, pp. 19–31, 2003.

[13] L. M. Shulman, "Gender differences in Parkinson's disease," *Gender Medicine*, vol. 4, no. 1, pp. 8–18, 2007.

[14] Y. Baba, J. D. Putzke, N. R. Whaley, Z. K. Wszolek, and R. J. Uitti, "Gender and the Parkinson's disease phenotype," *Journal of Neurology*, vol. 252, no. 10, pp. 1201–1205, 2005.

[15] K. P. Roland, J. M. Jakobi, C. Powell, and G. R. Jones, "Factors related to functional independence in females with Parkinson's disease: a systematic review," *Maturitas*, vol. 69, no. 4, pp. 304–311, 2011.

[16] C. E. Garber and J. H. Friedman, "Effects of fatigue on physical activity and function in patients with Parkinson's disease," *Neurology*, vol. 60, no. 7, pp. 1119–1124, 2003.

[17] J. Grace, A. Mendelsohn, and J. H. Friedman, "A comparison of fatigue measures in Parkinson's disease," *Parkinsonism and Related Disorders*, vol. 13, no. 7, pp. 443–445, 2007.

[18] M. Nallegowda, U. Singh, G. Handa et al., "Role of sensory input and muscle strength in maintenance of balance, gait, and posture in Parkinson's disease: a pilot study," *American Journal of Physical Medicine and Rehabilitation*, vol. 83, no. 12, pp. 898–908, 2004.

[19] M. D. Rothman, L. Leo-Summers, and T. M. Gill, "Prognostic significance of potential frailty criteria," *Journal of the American Geriatrics Society*, vol. 56, no. 12, pp. 2211–2216, 2008.

[20] B. Dijkstra, Y. Kamsma, and W. Zijlstra, "Detection of gait and postures using a miniaturised triaxial accelerometer-based system: accuracy in community-dwelling older adults," *Age and Ageing*, vol. 39, no. 2, Article ID afp249, pp. 259–262, 2010.

[21] J. L. Copeland and D. W. Esliger, "Accelerometer assessment of physical activity in active, healthy older adults," *Journal of Aging and Physical Activity*, vol. 17, no. 1, pp. 17–30, 2009.

[22] S. C. Webber and M. M. Porter, "Monitoring mobility in older adults using global positioning system (GPS) watches and accelerometers: a feasibility study," *Journal of Aging and Physical Activity*, vol. 17, no. 4, pp. 455–467, 2009.

[23] K. Karnik and D. J. Mazzatti, "Review of tools and technologies to assess multi-system functional impairment and frailty," *Clinical Medicine: Geriatrics*, vol. 2009, no. 3, pp. 1–8, 2009.

[24] H. L. Taylor, D. R. Jacobs, and B. Schucker, "A questionnaire for the assessment of leisure time physical activities," *Journal of Chronic Diseases*, vol. 31, no. 12, pp. 741–755, 1978.

[25] T. M. Gill, D. I. Baker, M. Gottschalk, P. N. Peduzzi, H. Allore, and A. Byers, "A program to prevent functional decline in physically frail, elderly persons who live at home," *New England Journal of Medicine*, vol. 347, no. 14, pp. 1068–1074, 2002.

[26] M. Fujikane, S. Katayama, K. Hirata, and N. Yokota, "Objective measurement of motor activity in Parkinson's disease by actigraphy. Clinical assessment of akinesia," *Nippon Rinsho*, vol. 55, no. 1, pp. 153–157, 1997.

[27] M. E. Morris, "Movement disorders in people with parkinson disease: a model for physical therapy," *Physical Therapy*, vol. 80, no. 6, pp. 578–597, 2000.

[28] M. J. Toth, P. S. Fishman, and E. T. Poehlman, "Free-living daily energy expenditure in patients with Parkinson's disease," *Neurology*, vol. 48, no. 1, pp. 88–91, 1997.

[29] Parkinson Society Canada (PSC), "Exercise for people with Parkinson," 2003, Retrieved from http://www.parkinson.ca/atf/cf/{9EBD08A9-7886-4B2D-A1C4-A131E7096BF8}/Exercise%20updated%20English%202010.pdf.

[30] C. Shaw, "Parkinson's disease exercise? Why not!," Vancouver Coastal Health, Catalogue # FM.490.P229.

[31] M. C. Morey, C. F. Pieper, G. M. Crowley, R. J. Sullivan, and C. M. Puglisi, "Exercise adherence and 10-year mortality in chronically Ill older adults," *Journal of the American Geriatrics Society*, vol. 50, no. 12, pp. 1929–1933, 2002.

[32] W. J. Chodzko-Zajko, D. N. Proctor, M. A. Fiatarone Singh et al., "Exercise and physical activity for older adults," *Medicine and Science in Sports and Exercise*, vol. 41, no. 7, pp. 1510–1530, 2009.

[33] N. Fallah, A. Mitnitski, S. D. Searle, E. A. Gahbauer, T. M. Gill, and K. Rockwood, "Transitions in frailty status in older adults in relation to mobility: a multistate modeling approach employing a deficit count," *Journal of the American Geriatrics Society*, vol. 59, no. 3, pp. 524–529, 2011.

[34] P. S. Baker, E. V. Bodner, and R. M. Allman, "Measuring life-space mobility in community-dwelling older adults," *Journal of the American Geriatrics Society*, vol. 51, no. 11, pp. 1610–1614, 2003.

[35] Q. L. Xue, L. P. Fried, T. A. Glass, A. Laffan, and P. H. M. Chaves, "Life-space constriction, development of frailty, and the competing risk of mortality: the women's health and aging study I," *American Journal of Epidemiology*, vol. 167, no. 2, pp. 240–248, 2008.

[36] K. Rockwood, R. A. Fox, P. Stolee, D. Robertson, and B. L. Beattie, "Frailty in elderly people: an evolving concept," *Canadian Medical Association Journal*, vol. 150, no. 4, pp. 489–495, 1994.

[37] D. Aarsland, J. P. Larsen, E. Tandberg, and K. Laake, "Predictors of nursing home placement in Parkinson's disease: a population-based, prospective study," *Journal of the American Geriatrics Society*, vol. 48, no. 8, pp. 938–942, 2000.

[38] A. Ashburn, E. Stack, R. M. Pickering, and C. D. Ward, "A community-dwelling sample of people with Parkinson's disease: characteristics of fallers and non-fallers," *Age and Ageing*, vol. 30, no. 1, pp. 47–52, 2001.

[39] J. S. Lou, G. Kearns, T. Benice, B. Oken, G. Sexton, and J. Nutt, "Levodopa improves physical fatigue in Parkinson's disease: a double-blind, placebo controlled, crossover study," *Movement Disorders*, vol. 18, no. 10, pp. 1108–1114, 2003.

[40] R. Shibley, H. J. Griffin, N. P. Quinn, and M. Jahanshahi, "Quality of life in Parkinson's disease: the relative importance of the symptoms," *Movement Disorders*, vol. 23, no. 10, pp. 1428–1434, 2008.

[41] J. A. Ávila-Funes, H. Amieva, P. Barberger-Gateau et al., "Cognitive impairment improves the predictive validity of the phenotype of frailty for adverse health outcomes: the three-city study," *Journal of the American Geriatrics Society*, vol. 57, no. 3, pp. 453–461, 2009.

[42] B. Scott, A. Borgman, H. Engler, B. Johnels, and S. M. Aquilonius, "Gender differences in Parkinson's disease symptom profile," *Acta Neurologica Scandinavica*, vol. 102, no. 1, pp. 37–43, 2000.

[43] M. C. Smith, H. Ellgring, and W. H. Oertel, "Sleep disturbances in Parkinson's disease patients and spouses," *Journal of the American Geriatrics Society*, vol. 45, no. 2, pp. 194–199, 1997.

[44] L. A. Lipsitz, "Physiological complexity, aging, and the path to frailty," *Science of Aging Knowledge Environment*, vol. 2004, no. 16, article pe16, 2004.

[45] N. N. Ahmed, S. J. Sherman, and D. VanWyck, "Frailty in Parkinson's disease and its clinical implications," *Parkinsonism and Related Disorders*, vol. 14, no. 4, pp. 334–337, 2008.

[46] F. Lauretani, M. Maggio, C. Silvestrini, A. Nardelli, M. Saccavini, and G. P. Ceda, "Parkinson's disease (PD) in the elderly: an example of geriatric syndrome (GS)?" *Archives of Gerontology and Geriatrics*, vol. 54, pp. 242–246, 2012.

[47] W. J. Mutch, A. Strudwick, S. K. Roy, and A. W. Downie, "Parkinson's disease: disability, review, and management," *British Medical Journal*, vol. 293, no. 6548, pp. 675–677, 1986.

Hindawi Publishing Corporation
Journal of Aging Research
Volume 2012, Article ID 135192, 7 pages
doi:10.1155/2012/135192

Research Article

Physical Activity among Veterans and Nonveterans with Diabetes

Erin D. Bouldin[1,2] and Gayle E. Reiber[1,2,3]

[1] *Health Services Research and Development Service, Department of Veterans Affairs, VA Puget Sound Health Care System, Seattle, WA 98101, USA*
[2] *Department of Epidemiology, University of Washington, Seattle, WA 98195, USA*
[3] *Department of Health Services, University of Washington, Seattle, WA 98195, USA*

Correspondence should be addressed to Erin D. Bouldin, erin.bouldin@va.gov

Received 18 February 2012; Accepted 17 May 2012

Academic Editor: Iris Reuter

Engaging in regular physical activity (PA), with or without a corresponding decrease in weight, is associated with improved health outcomes. The purpose of this study was to quantify the extent to which PA differed between veterans and nonveterans and to determine how diabetes and age influenced this association. Data from the 2009 Behavioral Risk Factor Surveillance System were used in this study. Respondents were classified as having diabetes if they reported ever being diagnosed with diabetes except during pregnancy. Respondents who reported ever serving on active military duty were classified as veterans. Based on self-report, we calculated the average minutes per week of moderate, vigorous, and total activity. After adjusting for sex, race and ethnicity, household income, education level, body mass index (BMI), and recent health checkup, veteran status was associated with a small but significantly larger amount of average weekly moderate PA (2.2 minutes, $P = 0.0058$) but not average weekly vigorous PA (-0.02 minutes, $P = 0.98$). Diabetes and prediabetes were associated with significantly lower mean levels of both moderate and vigorous intensity PA, as was increasing age. Consistent with prior research, veterans engaged in more PA than nonveterans. The association between diabetes, age, and physical activity did not differ by veteran status.

1. Introduction

Engaging in regular physical activity, with or without a corresponding decrease in weight, is associated with improved health outcomes among people with diabetes [1–4] and older adults [4–6]. Healthy People 2020 objectives include increasing glycemic, lipid, and blood pressure control among people with diabetes [7], all of which may be facilitated by increased physical activity [1]. Healthy People 2020 also includes an objective to increase the proportion of older adults with functional impairments who engage in leisure-time physical activity [7]. Guidelines from the National Institutes of Health [8] and the American College of Sports Medicine [9, 10] recommend that all adults, including those with diabetes, engage in regular moderate to vigorous physical activity. The 2008 Physical Activity Guidelines for Americans recognizes that any level of physical activity is beneficial, though the evidence is strongest for physical activity at or above the level of the recommended 150 minutes per week of moderate or 75 minutes per week of vigorous activity [4, 8]. They also recognize that older adults may have functional impairments that limit their ability to engage in physical activity, but recommend being as active as possible within safe limits [8]. Although physical activity is increasing among adults in the United States, only half of US adults meet these recommendations [11, 12]. Among older adults, the trend in physical activity is less clear [12], and roughly 40% of adults age 65 and older meet the physical activity guidelines [13].

Veterans in the United States are older, on average, than nonveterans [14] and are more likely than their nonveteran peers to meet physical activity guidelines [15]. Adults with diabetes are less likely to participate in regular exercise and less likely to meet recommendations than their peers without diabetes [16, 17]. Disability status, age, and concurrent health conditions are known to influence physical activity levels among adults [17–19], including among adults with diabetes [2, 18], but it is not clear whether veteran status influences these associations or whether veterans with diabetes and nonveterans with diabetes engage in different levels of physical activity. Therefore, the purpose of this study was to quantify the extent to which physical activity (PA) differed between veterans and nonveterans and to determine how diabetes and age influenced this association.

2. Methods

We used data from the Behavioral Risk Factor Surveillance System (BRFSS), a nationally representative cross-sectional telephone survey of noninstitutionalized adults in the United States and its territories coordinated by the Centers for Disease Control and Prevention (CDC), in this study. The BRFSS has demonstrated validity and reliability [20–22]. BRFSS respondents self-report their age. We categorized age into 18–34, 35–44, 45–54, 55–64, 65–74, and 75 and older in order to describe the sample, to calculate mean physical activity by age groups and veteran status, and to assess for effect modification. We used age as a continuous variable in the linear regression models. Respondents were classified as having diabetes if they reported ever being diagnosed with diabetes. Respondents who reported ever being told by their doctor that they have prediabetes or borderline diabetes were classified as having prediabetes. Women with gestational diabetes only and respondents who said they had never been told they have diabetes or pre-diabetes were classified as not having diabetes.

BRFSS respondents were asked about the amount and frequency of their weekly, nonwork moderate and vigorous intensity physical activity (PA). Moderate activities were defined as causing small increases in breathing or heart rate, while vigorous activities were defined as causing large increases in breathing or heart rate. We calculated the mean minutes of moderate, vigorous, and total (moderate plus vigorous) physical activity per week among respondents and also classified respondents as meeting or not meeting physical activity recommendations according to the 2008 Physical Activity Guidelines for Americans [8].

Respondents who reported ever serving on active duty in the military, National Guard, or reserve but who were not currently on active duty at the time of the survey were classified as veterans. Age, gender, educational attainment, race, ethnicity, and annual household income were used as categorical variables. Respondents were classified as having a disability if they reported an activity limitation or reported that they had a health problem requiring the use of special equipment (e.g., cane or wheelchair). We used the question "Would you say that in general your health is excellent, very good, good, fair, or poor?" to assess health-related quality of life and categorized responses into excellent/very good/good and fair/poor.

All measures used in this study were based on respondent self-report. In 2009, the BRFSS included 432,607 respondents; we excluded 70,469 respondents (16.3%) who were missing data on diabetes status, veteran status, age, sex, race/ethnicity, education, disability, time of most recent health checkup, body mass index (BMI), or average amount of moderate or vigorous physical activity per week. We retained respondents with missing income information but created a separate category for these respondents. Among our final study sample of 362,138, there were 43,048 (8.9%) who reported ever being diagnosed with diabetes, 5,678 who reported ever being diagnosed with pre-diabetes (1.2%), and 48,582 respondents (11.0%) who were veterans.

To describe the sample by veteran and diabetes status, we calculated proportions and Wald confidence intervals based on the binomial random variables. We used linear regression to model the mean frequency of moderate, vigorous, and total physical activity in an average week in three separate models. We report the β coefficients from these regression models, which represent the change in minutes of average physical activity per week associated with a one-unit change in the predictor. Because we assessed three physical activity outcomes, we used the Bonferroni correction for multiple comparisons and used an alpha value of 0.0167. We considered sex, race/ethnicity, household income, highest level of education, and body mass index (BMI) to be potential confounders based on previous studies [15–17]. The variables for recent health check-up, health insurance coverage, and having at least one personal health care provider were highly collinear, so we chose the single variable—recent health check-up—that best captured health service utilization since respondents who visited a health care professional may have been advised to engage in regular physical activity. Additionally, we were interested in whether veteran status modified the relationship between diabetes and physical activity or between age and physical activity and used an *a priori* P value of 0.05 to indicate statistical significance of the interaction. Although disability is associated with both diabetes and physical activity [18, 19], we considered disability to be in the causal pathway between diabetes and physical activity and therefore did not adjust for it. With the exception of the physical activity outcomes and age, all variables were categorical.

All analyses were conducted using SAS 9.2 with survey procedures and CDC-assigned weights to account for the complex sampling design of the BRFSS and to represent the US population by age, sex, and race/ethnicity. This study was reviewed as exempt by the VA Puget Sound Health Care System Institutional Review Board.

3. Results

Among veterans, the prevalence of diabetes was 15.4% (95% confidence interval [CI]: 14.9, 16.0), and the prevalence of pre-diabetes was 1.8% (95% CI: 1.5, 2.0; Table 1). Among nonveterans, the prevalence of diabetes and pre-diabetes was 8.0% (95% CI: 7.9, 8.2) and 1.2% (95% CI: 1.1, 1.2), respectively. Veterans were older than nonveterans (mean age 59.1 years [SE = 0.2] compared to 44.7 years [SE = 0.1]), and respondents with diabetes were older than respondents without diabetes (mean age with diabetes: 59.3 years [SE = 0.2], mean age with pre-diabetes: 55.6 [SE = 0.5], and mean age with no diabetes: 44.9 years [SE = 0.1]). More than 90% of veterans were male, while 44% of nonveterans were male. The majority of both Veterans and nonveterans

Table 1: Demographic and health characteristics of veterans and nonveterans in the United States, 2009 Behavioral Risk Factor Surveillance System.

Variable	Categories	Veterans weighted % (95% CI)	Nonveterans weighted % (95% CI)
Diabetes diagnosis	Diabetes	15.4 (14.9, 16.0)	8.0 (7.9, 8.2)
	Pre-diabetes	1.8 (1.5, 2.0)	1.2 (1.1, 1.2)
	No diabetes	82.8 (82.1, 83.4)	90.8 (90.6, 91.0)
Age	18–34	9.5 (8.7, 10.3)	32.5 (32.1, 32.9)
	35–44	12.4 (11.7, 13.1)	20.1 (19.8, 20.4)
	45–54	15.2 (14.6, 15.8)	19.9 (19.7, 20.2)
	55–64	23.2 (22.5, 23.8)	13.7 (13.5, 13.9)
	65–74	19.0 (18.5, 19.6)	7.7 (7.6, 7.9)
	75+	20.7 (20.1, 21.2)	5.9 (5.8, 6.0)
Sex	Male	91.8 (91.3, 92.3)	44.4 (44.0, 44.8)
Race, ethnicity	White, non-Hispanic	78.3 (77.4, 79.2)	61.7 (68.5, 69.3)
	Black, non-Hispanic	9.7 (9.0, 10.3)	9.7 (9.5, 9.9)
	Other race, non-Hispanic	5.2 (4.7, 5.6)	6.7 (6.5, 6.9)
	Any race, Hispanic	6.8 (6.1, 7.5)	14.7 (14.3, 15.0)
Highest level of education	Less than high school	5.0 (4.6, 5.3)	10.2 (10.0, 10.5)
	High school degree or equivalent	28.3 (27.5, 29.1)	27.4 (27.1, 27.7)
	Some college	31.2 (30.3, 32.0)	26.5 (26.2, 26.8)
	College or beyond	35.5 (34.7, 36.3)	35.8 (35.5, 36.2)
Annual household income	< $15,000	4.9 (4.5, 5.2)	9.2 (9.0, 9.5)
	$15,000–$24,999	13.2 (12.6, 13.8)	13.7 (13.5, 14.0)
	$25,000–$49,999	26.9 (26.1, 27.6)	21.7 (21.4, 22.0)
	$50,000–$74,999	17.7 (17.1, 18.4)	14.4 (14.1, 14.7)
	≥ $75,000	28.6 (27.8, 29.4)	30.2 (29.8, 30.5)
	Missing	8.9 (8.2, 9.2)	10.8 (10.5, 11.0)
Disability status	Disability	29.9 (29.1, 30.7)	19.4 (19.2, 19.7)
General health rating	Fair or poor	19.2 (18.5, 19.8)	14.9 (14.6, 15.1)
Body mass index (BMI)	Ideal (<25.0)	25.4 (24.6, 26.2)	37.4 (37.1, 37.8)
	Overweight (25.0–29.9)	44.7 (43.8, 45.5)	35.2 (34.8, 35.5)
	Obese (≥30.0)	29.9 (29.1, 30.7)	27.4 (27.0, 27.7)
Health insurance coverage	Any (including government/VA)	91.7 (91.0, 92.3)	84.7 (84.3, 85.0)
Health care cost	Time in past 12 months when could not see doctor because of cost	8.3 (7.7, 8.9)	15.2 (14.9, 15.5)
Health provider	No personal doctor	13.3 (12.5, 14.0)	18.9 (18.6, 19.3)
Last health check-up	Within past year	77.4 (76.6, 78.2)	67.1 (66.8, 67.5)
	1–5 years ago	16.4 (15.7, 17.1)	23.6 (23.2, 23.9)
	Never	6.2 (5.8, 6.6)	9.3 (9.0, 9.5)

CI: confidence interval.

reported their race and ethnicity as white, non-Hispanic, though veterans were more likely to be so than nonveterans (78.3% of veterans and 61.7% of nonveterans reported white, non-Hispanic race/ethnicity). Veterans and nonveterans had similar levels of education and annual household income. Disability was significantly more common among veterans: 29.9% of veterans were classified as having a disability compared to 19.4% of nonveterans. Veterans tended to have higher BMI than nonveterans and were more frequently classified as overweight (25.4% of Veterans and 37.4% of nonveterans were classified as ideal weight based on their BMI, and 44.7% of veterans compared to 35.2% of nonveterans were overweight). Veterans were more likely than nonveterans to have any form of health insurance (91.7% versus 84.7%) and less likely to report that cost had been a barrier to accessing health care when needed within the past year (8.3% compared to 15.2%). Veterans also were less likely to report that they did not have a personal doctor (13.3%

Table 2: Mean minutes of moderate, vigorous, and total physical activity in an average week reported by veterans and nonveterans with and without diabetes in the United States, 2009 Behavioral Risk Factor Surveillance System.

Demographic group	Moderate mean (SE)	Vigorous mean (SE)	Total mean (SE)
Veterans (n = 48,582)	57.2 (0.6)	35.3 (0.5)	92.5 (0.9)
With diabetes (n = 8,515)	48.7 (1.3)	24.4 (1.3)	73.1 (2.0)
With pre-diabetes (n = 947)	48.5 (3.1)	29.7 (2.8)	78.1 (4.9)
Without diabetes (n = 39,120)	59.0 (0.7)	37.4 (0.6)	96.5 (1.1)
Age 18–54 (n = 10,471)	60.4 (1.3)	42.7 (1.1)	103.2 (2.0)
Age 55–64 (n = 12,664)	57.5 (1.0)	36.1 (1.1)	93.6 (1.7)
Age 65–74 (n = 12,507)	58.3 (1.0)	31.5 (0.8)	89.9 (1.5)
Age ≥ 75 (n = 12,940)	50.2 (1.1)	24.5 (0.8)	74.6 (1.5)
Nonveterans (n = 313,556)	53.3 (0.3)	33.2 (0.2)	86.5 (0.4)
With diabetes (n = 34,533)	42.8 (0.7)	19.0 (0.5)	61.8 (1.0)
With pre-diabetes (n = 4,731)	51.3 (3.0)	26.4 (1.7)	77.7 (4.2)
Without diabetes (n = 274,292)	54.3 (0.3)	34.5 (0.3)	88.8 (0.5)
Age 18–54 (n = 158,990)	55.1 (0.4)	37.3 (0.3)	92.4 (0.6)
Age 55–64 (n = 68,919)	50.6 (0.5)	26.4 (0.4)	76.9 (0.7)
Age 65–74 (n = 49,040)	50.8 (0.5)	21.9 (0.4)	72.7 (0.7)
Age ≥ 75 (n = 36,607)	40.8 (0.6)	13.6 (0.4)	54.5 (0.8)

SE: standard error.

versus 18.9%) and were more likely to have had a health check-up within the past year (77.4% compared to 67.1%).

The mean amount of moderate physical activity (PA) among veterans was 57.2 minutes per week (standard error [SE] = 0.6), and the mean amount of vigorous physical activity was 35.3 minutes per week (SE = 0.5) for a total mean of 92.5 minutes of physical activity weekly (SE = 0.9; Table 2). The mean minutes of moderate, vigorous, and total PA were 53.3 (SE = 0.3), 33.2 (SE = 0.2), and 86.5 (SE = 0.4) per week, respectively, among nonveterans. Regardless of veteran status, people with diabetes engaged in less moderate PA and vigorous PA per week than people without diabetes. Among veterans with diabetes, the mean moderate, vigorous, and total PA amounts per week were 48.7 (SE = 1.3) minutes, 24.4 (SE = 1.3) minutes, and 73.1 (SE = 2.0) minutes, respectively, while among veterans without diabetes, the means were 59.0 (SE = 0.7) minutes of moderate PA, 37.4 (SE = 0.6) minutes of vigorous PA, and 96.5 (SE = 1.1) minutes of total PA weekly. Veterans with pre-diabetes averaged 48.5 (SE = 3.1) minutes of moderate PA, 29.7 (SE = 2.8) minutes of vigorous PA, and 78.1 (SE = 4.9) minutes of total PA weekly. Among nonveterans with diabetes, mean moderate PA was 42.8 (SE = 0.7) minutes per week, mean vigorous PA was 19.0 (SE = 0.5) minutes per week, and mean total PA was 61.8 (SE = 1.0) minutes per week compared to 54.3 (SE = 0.3) minutes, 34.5 (SE = 0.3) minutes, and 88.8 (SE = 0.5) minutes, respectively, among nonveterans without diabetes. Nonveterans with pre-diabetes averaged 51.3 (SE = 3.0) minutes of moderate PA, 26.4 (SE = 1.7) minutes of vigorous PA, and 77.7 (SE = 4.2) minutes of total PA weekly. Among both veterans and nonveterans, respondents with diabetes had a larger proportion of their total physical activity from moderate physical activity (66.6% of PA among veterans with diabetes, 62.1% of PA among veterans with prediabetes, 69.3% among nonveterans with diabetes, 66.0% among nonveterans with prediabetes, and 61.1% of PA among veterans and nonveterans without diabetes was moderate intensity). Among both veterans and nonveterans, total physical activity decreased with age, with much of the decrease related to a decrease in vigorous activity. Veterans age 18–54 averaged 60.4 (SE = 1.3) minutes of moderate, 42.7 (SE = 1.1) minutes of vigorous, and 103.2 (SE = 2.0) minutes of total PA weekly. Among veterans age 75 and older, the averages were 50.2 (SE = 1.1) minutes of moderate, 24.5 (SE = 0.8) minutes of vigorous, and 74.6 (SE = 1.5) minutes of total PA per week. Nonveterans age 18–54 averaged 55.1 (SE = 0.4) minutes of moderate, 37.3 (SE = 0.3) minutes of vigorous, and 92.4 (SE = 0.6) minutes of total PA weekly. Among nonveterans age 75 and older, the averages were 40.8 (SE = 0.6) minutes of moderate, 13.6 (SE = 0.4) minutes of vigorous, and 54.5 (SE = 0.8) minutes of total PA per week.

When physical activity was categorized based on the Physical Activity Guidelines for Americans recommendations, nearly half of veterans and nonveterans met PA recommendations (48.7% and 49.3%, resp.). Additionally, 37.1% of veterans and 38.3% of nonveterans engaged in some physical activity in an average week but did not meet recommendations, and 14.1% of veterans and 12.3% of nonveterans engaged in no physical activity.

We adjusted each linear regression model for sex, race and ethnicity, household income, education level, BMI category, and recent health check-up. In the fully adjusted models (Table 3), we found no evidence of effect modification by veteran status ($P = 0.07$ for diabetes and $P = 0.89$ for age in categories) so report results adjusted by veteran status rather than stratified.

TABLE 3: Adjusted* estimated differences in mean minutes of moderate, vigorous, and total physical activity (PA) per week among veterans and nonveterans in the United States, 2009 Behavioral Risk Factor Surveillance System.

Variable	Category	Moderate PA minutes (β) (95% CI)	Vigorous PA minutes (β) (95% CI)	Total† PA minutes (β) (95% CI)
Veteran status	Veteran	2.2 (0.6, 3.8)	−0.02 (−1.3, 1.3)	2.2 (−0.1, 4.5)
	Nonveteran	Reference	Reference	Reference
Diagnosed diabetes status	Diabetes	−8.0 (−9.4, −6.5)	−7.2 (−8.3, −6.1)	−15.2 (−17.3, −13.1)
	Pre-diabetes	−1.3 (−6.3, 3.7)	−1.5 (−4.4, 1.3)	−2.8 (−9.8, 4.2)
	Neither diabetes nor pre-diabetes	Reference	Reference	Reference
Age	One-year increase	−0.1 (−0.2, −0.1)	−0.3 (−0.4, −0.3)	−0.5 (−0.5, −0.4)

*Adjusted for sex, race/ethnicity, education, household income, body mass index, and last health check-up.
†Total physical activity is the sum of moderate and vigorous physical activity.
β: linear regression coefficient, indicating the minutes per week associated with a one-unit change in the exposure variable.
CI: confidence interval.

In the adjusted moderate PA model, veterans engaged in a small but statistically significantly higher amount of PA on average (2.2 minutes; 95% CI: 0.6, 3.8; $P = 0.0058$). Respondents with diabetes averaged 8.0 (95% CI: −9.4, −6.5; $P < 0.0001$) fewer minutes, and respondents with pre-diabetes averaged 1.3 fewer minutes, (95% CI: −6.3, 3.7; $P = 0.62$) of moderate PA weekly than respondents without diabetes after adjusting for covariates. For each one year increase in age, respondents averaged 0.1 fewer minutes (95% CI: −0.2, −0.1; $P < 0.001$) of moderate PA weekly, adjusted for covariates.

In the adjusted vigorous PA model, there was no statistically significant difference in average weekly PA by veteran status (−0.02 minutes; 95% CI: −1.3, 1.3; $P = 0.98$). Respondents with diabetes averaged 7.2 (95% CI: −8.3, −6.1; $P < 0.0001$) fewer minutes and respondents with pre-diabetes averaged 1.5 fewer minutes (95% CI: −4.4, 1.3; $P = 0.30$) of vigorous PA weekly than respondents without diabetes after adjusting for covariates. For each one year increase in age, respondents averaged 0.3 fewer minutes (95% CI: −0.4, −0.3; $P < 0.001$) of vigorous PA weekly, adjusted for covariates.

In both models, women averaged significantly less weekly PA than men (data not shown; available upon request). Respondents who reported Hispanic ethnicity had a small but statistically significantly lower mean amount of moderate PA weekly compared to white, non-Hispanic respondents. For other categories of race and ethnicity, the trend was not statistically significant in either model. People with lower levels of education had significantly higher weekly PA than people with a college degree or higher, on average. The relationship between income and PA differed by the type of PA: for moderate PA, people with lower income tended to have higher mean PA, while for vigorous PA, people with lower income had a higher mean PA. Across both moderate PA and vigorous PA models, overweight and obesity were associated with a significantly lower amount of PA per week, on average.

4. Discussion

We identified a strong and statistically significant association between diabetes and physical activity and between age and physical activity among both veterans and nonveterans. The association between pre-diabetes and physical activity was much weaker. The demographic profile of veterans in this study was similar to a previous analysis of BRFSS data and VA records [23]. Consistent with the limited prior research on the topic [15], veterans engaged in more physical activity than their nonveteran peers. As suggested by Littman et al. [15], this may be a result of their fitness for military service or a result of the tendency to continue physical activity after the high level of activity associated with military training and service. Factors associated with lower physical activity were consistent across veterans and nonveterans.

We classified respondents who had been told by a doctor that they had pre-diabetes or borderline diabetes as a separate category in this study. These individuals likely had impaired fasting glucose or glucose tolerance or high hemoglobin A1c [24] and are therefore at higher risk for developing diabetes and for poor cardiovascular health [24, 25]. In this study, respondents with pre-diabetes had, on average, higher levels of physical activity and lower levels of disability than respondents with full-blown diabetes; including these respondents in the group with diabetes would have attenuated differences between respondents with and without diabetes. Diabetes and pre-diabetes are known to be underdiagnosed: according to an analysis of National Health and Nutrition Examination Survey (NHANES) data by Cowie et al. [26], 19.0% of adults had undiagnosed diabetes, and an additional 3.5% were at high risk for diabetes based on their hemoglobin A1c. In a BRFSS reliability study, Martin and colleagues found that the sensitivity of BRFSS diabetes question had moderate sensitivity and high specificity compared to medical record review [22]. The underdiagnosis of diabetes and pre-diabetes and the moderate sensitivity of the diabetes question used in this study likely led to misclassification in this study which may have resulted in underestimating the association between diabetes or pre-diabetes and physical activity.

Data from the BRFSS are designed to represent the non-institutionalized adult population; therefore, these results are generalizable to the community-dwelling residents of the United States and its territories who age 18 and older. These data are cross-sectional, so the temporal sequence of the associations reported is unclear. It may not be true that veteran status or diabetes causes people to be more or less physically active. Nonetheless, there is a public health benefit to quantifying the amount of PA among people with diabetes, and the relationship between diabetes, pre-diabetes, and physical activity identified in this study may be useful to health care providers advising patients. Likewise, because of the cross-sectional nature of the data, we cannot be sure from this study that aging causes a decline in physical activity since individuals were not followed over time. However, given the body of existing research on the topic, we feel the decline in activity and the capacity for exercise associated with aging has been well established [4, 27, 28].

All data in this study were self-reported and therefore are subject to recall and social desirability biases. Based on data from the 2003-2004 NHANES, in which height and weight were measured directly and accelerometers were used to capture physical activity information, the proportion of US adults who meet physical activity guidelines was below 5% [29], much lower than the 50% estimate in this study and others (e.g., [15, 17]) obtained from self-reported data. This overreporting may result in nondifferential misclassification (if all respondents overreported their physical activity). It is possible that a differential social desirability bias exists since respondents with diabetes may have received physician advice to exercise more often than adults without diabetes. For these reasons, the difference in estimates between groups in this study may be more appropriate than treating the means or proportions themselves as accurate reflections of physical activity among veterans and people with diabetes in the general population.

Although the estimated differences in physical activity minutes per week were small, they are meaningful in terms of health outcomes. For example, Buman et al. [30] found that replacing 30 minutes of sedentary behavior each day with light intensity physical activity was associated with improved physical health among older adults; this benefit increased as the intensity of activity increased. Likewise, Healy et al. [31] found that light intensity physical activity was associated with improved glucose control, as measured by the two-hour postchallenge plasma glucose but not fasting glucose. Additional research is needed to understand whether self-reported moderate physical activity includes light intensity physical activity, particularly among older adults [32], including veterans and individuals with diabetes [33]. Such research may include an assessment of whether additional categories of physical activity should be routinely added to health surveys like the BRFSS.

Disclosure

The views expressed in this paper are those of the authors and do not necessarily reflect the position or policy of the Department of Veterans Affairs.

Acknowledgments

This paper is based upon work in part by the U.S. Department of Veterans Affairs, Office of Research and Development, Health Services Research and Development, and Dr. Reiber's Research Career Scientist Award RCS no. 98-353.

References

[1] American Diabetes Association, "Standards of medical care in diabetes-2011," *Diabetes Care*, vol. 34, supplement 1, pp. S11–S61, 2011.

[2] S. R. Colberg, R. J. Sigal, B. Fernhall et al., "Exercise and type 2 diabetes: the American College Of Sports Medicine and The American Diabetes Association: joint position statement executive summary," *Diabetes Care*, vol. 33, no. 12, pp. 2692–2696, 2010.

[3] S. Mora, I. M. Lee, J. E. Buring, and P. M. Ridker, "Association of physical activity and body mass index with novel and traditional cardiovascular biomarkers in women," *Journal of the American Medical Association*, vol. 295, no. 12, pp. 1412–1419, 2006.

[4] Physical Activity Guidelines Advisory Committee, *Physical Activity Guidelines Advisory Committee Report, 2008*, US Department of Health and Human Services, Washington, DC, USA, 2008.

[5] I. A. Lang, J. M. Guralnik, and D. Melzer, "Physical activity in middle-aged adults reduces risks of functional impairment independent of its effect on weight," *Journal of the American Geriatrics Society*, vol. 55, no. 11, pp. 1836–1841, 2007.

[6] D. R. Berk, H. B. Hubert, and J. F. Fries, "Associations of changes in exercise level with subsequent disability among seniors: a 16-year longitudinal study," *Journals of Gerontology A*, vol. 61, no. 1, pp. 97–102, 2006.

[7] Healthy People, Office of Disease Prevention and Health Promotion, and United States Department of Health and Human Services, DC. Healthy People 2020, http://www.healthypeople.gov/.

[8] United States Department of Health and Human Services, Physical Activity Guidelines for Americans, 2008, http://www.health.gov/paguidelines/.

[9] W. L. Haskell, I. M. Lee, R. R. Pate et al., "Physical activity and public health: updated recommendation for adults from the American College of Sports Medicine and the American Heart Association," *Circulation*, vol. 116, no. 9, pp. 1081–1093, 2007.

[10] M. E. Nelson, W. J. Rejeski, S. N. Blair et al., "Physical activity and public health in older adults: recommendation from the American College of Sports Medicine and the American Heart Association," *Circulation*, vol. 116, no. 9, pp. 1094–1105, 2007.

[11] J. Kruger, H. W. Kohl, and I. J. Miles, "Prevalence of regular physical activity among adults-United States, 2001 and 2005," *Morbidity and Mortality Weekly Report*, vol. 56, no. 46, pp. 1209–1212, 2007.

[12] A. G. Knuth and P. C. Hallal, "Temporal trends in physical activity: a systematic review," *Journal of Physical Activity and Health*, vol. 6, no. 5, pp. 548–559, 2009.

[13] "CDC Web-Enabled Analysis Tool: cross-tabulation of age (18–64, 65+) and physical activity (Meets recommendation for moderate or vigorous physical activity) using 2009 national BRFSS data," 2012, http://apps.nccd.cdc.gov/s_broker/WEAT-SQL.exe/weat/freq_year.hsql.

[14] US Census Bureau, "Sex by age by Veteran status for the civilian population 18 years and over," American Community Survey 1-year estimates, 2010, http://factfinder2.census.gov/faces/tableservices/jsf/pages/productview.xhtml?pid=ACS_10_1YR_B21001&prodType=table.

[15] A. J. Littman, C. W. Forsberg, and T. D. Koepsell, "Physical activity in a national sample of veterans," *Medicine and Science in Sports and Exercise*, vol. 41, no. 5, pp. 1006–1013, 2009.

[16] E. H. Morrato, J. O. Hill, H. R. Wyatt, V. Ghushchyan, and P. W. Sullivan, "Physical activity in U.S. adults with diabetes and at risk for developing diabetes, 2003," *Diabetes Care*, vol. 30, no. 2, pp. 203–209, 2007.

[17] G. Zhao, E. S. Ford, C. Li, and L. S. Balluz, "Physical activity in U.S. older adults with diabetes mellitus: prevalence and correlates of meeting physical activity recommendations," *Journal of the American Geriatrics Society*, vol. 59, no. 1, pp. 132–137, 2011.

[18] S. E. Boslaugh and E. M. Andresen, "Correlates of physical activity for adults with disability," *Preventing Chronic Disease*, vol. 3, no. 3, p. A78, 2006.

[19] J. H. Rimmer, L. A. Wolf, B. S. Armour, and L. B. Sinclair, "Physical activity among adults with a disability-United States, 2005," *Morbidity and Mortality Weekly Report*, vol. 56, no. 39, pp. 1021–1024, 2007.

[20] E. M. Gentry, W. D. Kalsbeek, and G. C. Hogelin, "The behavioral risk factor surveys: II. Design, methods, and estimates from combined state data," *American Journal of Preventive Medicine*, vol. 1, no. 6, pp. 9–14, 1985.

[21] P. L. Remington, M. Y. Smith, D. F. Williamson, R. F. Anda, E. M. Gentry, and G. C. Hogelin, "Design, characteristics, and usefulness of state-based behavioral risk factor surveillance: 1981–87," *Public Health Reports*, vol. 103, no. 4, pp. 366–375, 1988.

[22] L. M. Martin, M. Leff, N. Calonge, C. Garrett, and D. E. Nelson, "Validation of self-reported chronic conditions and health services in a managed care population," *American Journal of Preventive Medicine*, vol. 18, no. 3, pp. 215–218, 2000.

[23] G. E. Reiber, T. D. Koepsell, C. Maynard, L. B. Haas, and E. J. Boyko, "Diabetes in Nonveterans, Veterans, and Veterans Receiving Department of Veterans Affairs Health Care," *Diabetes Care*, vol. 27, supplement 2, pp. B3–B9, 2004.

[24] American Diabetes Association, "Diagnosis and classification of diabetes mellitus," *Diabetes Care*, vol. 33, supplement 1, pp. S62–S69, 2010.

[25] S. Y. Rhee and J. T. Woo, "The prediabetic period: review of clinical aspects," *Diabetes and Metabolism Journal*, vol. 35, no. 2, pp. 107–116, 2011.

[26] C. C. Cowie, K. F. Rust, D. D. Byrd-Holt et al., "Prevalence of diabetes and high risk for diabetes using A1C criteria in the U.S. population in 1988-2006," *Diabetes Care*, vol. 33, no. 3, pp. 562–568, 2010.

[27] American College of Sports Medicine, "American college of sports medicine position stand: exercise and physical activity for older adults," *Medicine and Science in Sports and Exercise*, vol. 41, no. 7, pp. 1510–1530, 2009.

[28] S. A. Hawkins and R. A. Wiswell, "Rate and mechanism of maximal oxygen consumption decline with aging: implications for exercise training," *Sports Medicine*, vol. 33, no. 12, pp. 877–888, 2003.

[29] R. P. Troiano, D. Berrigan, K. W. Dodd, L. C. Mâsse, T. Tilert, and M. Mcdowell, "Physical activity in the United States measured by accelerometer," *Medicine and Science in Sports and Exercise*, vol. 40, no. 1, pp. 181–188, 2008.

[30] M. P. Buman, E. B. Hekler, W. L. Haskell et al., "Objective light-intensity physical activity associations with rated health in older adults," *American Journal of Epidemiology*, vol. 172, no. 10, pp. 1155–1165, 2010.

[31] G. N. Healy, D. W. Dunstan, J. Salmon et al., "Objectively measured light-intensity physical activity is independently associated with 2-h plasma glucose," *Diabetes Care*, vol. 30, no. 6, pp. 1384–1389, 2007.

[32] T. Prohaska, E. Belansky, B. Belza et al., "Physical activity, public health, and aging: critical issues and research priorities," *Journals of Gerontology B*, vol. 61, no. 5, pp. S267–S273, 2006.

[33] D. E. Rosenberg, C. H. Bombardier, J. M. Hoffman, and B. Belza, "Physical activity among persons aging with mobility disabilities: shaping a research agenda," *Journal of Aging Research*, vol. 2011, Article ID 708510, 16 pages, 2011.

Hindawi Publishing Corporation
Journal of Aging Research
Volume 2012, Article ID 612918, 7 pages
doi:10.1155/2012/612918

Research Article

"Convivência" Groups: Building Active and Healthy Communities of Older Adults in Brazil

Tânia R. Bertoldo Benedetti,[1] Eleonora d'Orsi,[2] Andiara Schwingel,[3] and Wojtek J. Chodzko-Zajko[4]

[1] *Department of Physical Education, Center of Sports, Federal University of Santa Catarina, Campus Universitário Reitor João David Ferreria Lima, Caixa Postal 476, Trindade, 88040-900 Florianópolis, SC, Brazil*
[2] *Department of Public Health, Center of Health Science, Federal University of Santa Catarina, Campus Universitário Reitor João David Ferreria Lima, Caixa Postal 476, Trindade, 88040-900 Florianópolis, SC, Brazil*
[3] *Department of Kinesiology and Community Health, University of Illinois at Urbana-Champaign, 212A Huff Hall MC-588, 1206 South Fourth Street, Champaign, IL 61820, USA*
[4] *Department of Kinesiology and Community Health, University of Illinois at Urbana-Champaign, Louise Freer Hall, 906 S. Goodwin Avenue, Urbana, IL 61801, USA*

Correspondence should be addressed to Tânia R. Bertoldo Benedetti, benedetti@cds.ufsc.br

Received 15 February 2012; Revised 9 April 2012; Accepted 23 April 2012

Academic Editor: Lynn Rochester

In old age, social groups can be a crucial component for health and well-being. In 2009-2010, a follow-up survey was carried out in Florianópolis, Brazil to understand the impact of a variety of programs established since 2002 that were designed to enhance social activities among the older adult population. This study employed two surveys within the population of older adults in Florianópolis. The first survey interviewed a total of 875 older adults in 2002, and the second survey involved 1,705 older adults between 2009 and 2010. By 2010, many new programs were offered in the community and the enrollment of older adults in social programs followed similar trends. "Convivência" groups stood out as extremely popular social groups among this population. This paper discusses some of the potential outcomes associated with participation in "convivência" groups.

1. Introduction

Regular engagement in social activities is an important component of successful aging [1]. Having family and friends to spend time with helps people to find their social identity and purpose in life. Social activity starts with the family at home and goes beyond to schools, work, clubs, community, and faith-oriented groups. These opportunities are very important to people's well-being and quality of life [2, 3].

Social groups are examples of structured opportunities for people that share similar interests to get together. These interests could range from religion, politics, sports and leisure, and education, among others [4]. Although there is no conclusive evidence of causality, a positive association between social group participation and good health is found in the literature. Social groups are important to people and they play different roles throughout the life course. Older adults engaged in social groups share better physical and mental health than their counterparts who are not engaged [5–9]. Studies show that on average, socially engaged older adults have less depression [6, 10], live more independently [8, 11], have better physical and cognitive functioning [12], have higher levels of life satisfaction [9, 13], and are more likely to be engaged in healthy lifestyles [14–17].

Studies conducted in Brazil have reported that only a small proportion of older adults regularly participate in social groups [18, 19]. Some barriers that prevent older Brazilians from engaging in social groups are meetings held in inconvenient locations, lack of time due to family caregiving roles, and lack of company to motivate regular participation [20].

Efforts to increase social participation among older adults have been initiated worldwide. In the early 1990s, Brazil initiated a program from city halls and faith-based organizations to increase the social engagement of older adults. Public efforts were devoted to the creation of community-based social groups known as "convivência" groups. This initiative was financially supported by the Brazilian National Public Policy for Older Adults in 2003 [21] and was consistent with the strategies identified in the 2002 World Assembly on Ageing held in Madrid, Spain [22].

"Convivência" groups were designed to promote social exchanges among older adults. This is an effort grounded in public health that aims to promote active aging by encouraging healthy lifestyles, independence, productivity, and participation in civic activities [23]. The structure of "convivência" groups involves a daily three-hour period in the afternoon during which a number of different leisure and educational activities are made available to participating older adults aged 60+. Among the most popular activities are folk dance, bingo, seminars about health, exercise classes, choirs, ballroom dance, art craft activities, and watching theater and/or dance performances. Meetings are held in church halls and community centers, and have a participation of 30 to 100 people per day [24, 25].

"Convivência" groups are part of a national program for older adults in Brazil. There have been some studies to document the benefits of this initiative in different parts of the country [26]. In 2002, a comprehensive survey was conducted in southern Brazil which revealed that 20% of older adults were participating regularly in "convivência" groups. The majority of participants were women of low income and with little education [20, 25, 27]. A positive association was found between participating in "convivência" groups and participating in physical activities. Older adults engaged in "convivência" groups were found to be more physically active than their counterparts who were not engaged. Findings of this study were disseminated by the local media and through publications [28]. As a result, the municipality of Florianópolis launched in 2006 a program called "*Capital do Idoso*." This ongoing program was developed to improve public health in four target areas of intervention: prevention, promotion, therapy, and rehabilitation.

In 2009-2010, a follow-up survey was conducted to understand the impact of programs on social engagement among the older adult population [29]. The changes, benefits, and challenges of these public efforts for the older adult population and for the city as a whole are documented in this paper.

2. Methods

This paper was based on two surveys conducted in Florianópolis, capital of Santa Catarina State, Southern Brazil. Florianópolis is a middle size city with approximately 421,203 residents [30]. The first survey was conducted in 2002, and the second in 2010 [28, 29]. The first survey interviewed a total of 875 older adults (437 men and 438 women) with average age of 71.6 ± 7.9 years. In 2002, the population of older adults represented 8.4% of the total population. The data were collected by the research team from September to December 2002. This research was approved by the Ethics Committee for Research on Human Beings of the Universidade Federal de Santa Catarina, Brazil (Protocol no. 051/2001).

The second survey was conducted between 2009 and 2010 with 1,705 older adults (616 men and 1089 women). In 2010, the population of older adults in Florianópolis represented 11% of the total population [30]. The average age of the study participants was 70.7 ± 8.0 years. The data were collected by the research team between September 2009 and June 2010. The research was approved by the Ethics Committee for Research on Human Beings of the Universidade Federal de Santa Catarina, Brazil (Protocol no. 352/2008). A statement of informed consent was obtained from each participant prior the initiation of data collection. Trained interviewers conducted all interviews. The average time taken to conduct each in-home interview was around 60 minutes.

A Brazilian national database was used to select a representative sample of older persons (Instituto Brasileiro de Geografia e Estatística (IBGE)). Both 2002 and 2009 surveys used randomized census tracts. Each census tract has about 300–350 homes. In 2002, the research team interviewed one older adult man and one woman per census tract, whereas the second survey selected census tracts and homes at random.

2.1. Research Instruments. A comprehensive questionnaire was administered to each participant. The questionnaire in survey one included demographic information, physical health, the use of medical and dental services, activities of daily living and falls, physical activity levels, social resources, socioeconomic status, mental health, and needs and issues that affect older adults lives. Survey two also included questions about lifestyle, women's health, eating habits, mobility, functional capacity, environmental opportunities for physical activity, and elder abuse. For the purpose of this paper we report only demographic information, socioeconomic status, and social resources.

The International Physical Activity Questionnaire (IPAQ-long form) was used to assess physical activity levels [31]. The IPAQ was developed as a relatively simple self-report instrument that would be available in many languages and which would enable researchers to estimate physical activity levels in different countries and compare these data. A key feature of the IPAQ questionnaire is its ability to provide, in detail, participation estimates for multiple domains of physical activity, including leisure time physical activity, physical activity for transportation, physical activity in the home, and physical activity at work. Although the IPAQ explores physical activity levels in four domains, in 2010 we chose to use data only from two domains, transportation and leisure time physical activity. For the purpose of this paper, the middle level proposed in the original IPAQ was suppressed, following recommendations from previous surveys using IPAQ with older adults in Brazil [32, 33]. Therefore, older adults who carried out moderate or vigorous physical activities within the four domains for 150 minutes per week or over were classified as more active, whereas those who did not reach 150 minutes per week were classified as less active [31].

Analysis of variance was used to examine differences between surveys for the continuous variables. Adjustments for age, sex, education, and income differences were performed using analysis of covariance. Chi-squared tests were used for the analyses of categorical variables among the surveys. All statistical analyses were performed using SPSS 19.0 for Windows (SPSS Inc., Chicago, IL, USA) and statistical significance was set at $P < 0.05$.

3. Results

Table 1 shows the distribution of older adults by gender and socioeconomic status of the two surveys conducted in 2002, and in 2010. The average age of participants was not significantly different between the two surveys. In 2002, it was 71.5 years, and 70.6 years in 2010. The first survey included 50% women, whereas in 2010 they comprised 64% of the sample. In both surveys, the great majority of older adults was married and had less than 8 years of education. Household income source was mainly from public retirement pensions. A decline in overall household income was observed between years 2002 and 2010. Households with higher income levels decreased from 20% in 2002 to 10% in 2010. Illiterate older adults were the single largest socioeconomic group.

Table 2 shows participation of the older adults in "convivência groups", between 2002 and 2010.

Table 3 shows a list of public programs available to all older adults in Florianópolis city, between years 2002 and 2010. Data collected in 2002 has been published elsewhere [34]. Data from both surveys shows a clear increase in opportunities for social activities among older adults in Florianópolis from 2002 to 2010. By 2010, many new programs that were offered to the community and the enrollment of older adults had also increased. For instance, between 2002 and 2010, the total increase in "convivência" group members was 6,849 users. This outnumbers the 5,972 new users for the all other non-"convivência" physical activity, education, and dance groups offered in the city. "Convivência" groups are viewed as extremely popular social groups among older adults in Florianópolis. Adding totals from Table 3, there were 14,849 reported users of the "convivência" groups in 2010, and 9,388 enrolled in other non-"convivência" programs.

The diversity of types of programs also increased. By 2010, activities such as Pilates, "*bailes*" (ballroom dance), and other physical activities had become very popular. In addition, in the 2010 survey, our data suggest that greater attention was paid by health professionals to the promotion of healthy lifestyles and disease management.

In order to understand the influence of engagement in social programs, we examined changes in social activity over the time course of the study (Figure 1). Data from year 2010 show a significant increase in participation in "convivência" groups when compared with year 2002; reflecting a greater percentage of older adults engaged in social activities with friends. One of the main goals of the "convivência" groups

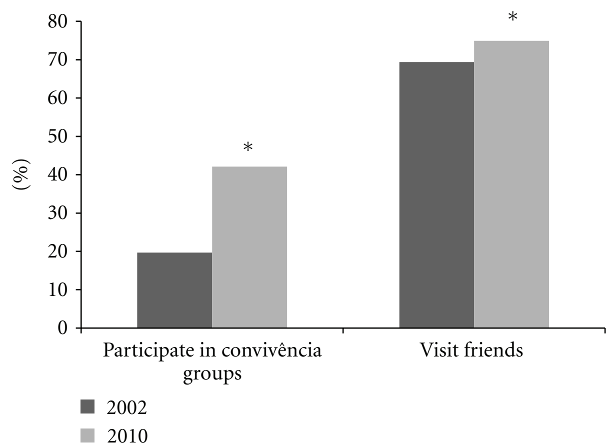


Figure 1: Participation in "convivência" groups and visits to friends between 2002 and 2010.

is to engage local residents of same cohort in social activities and therefore increase and strengthen their circle of friends.

To increase our understanding about lifestyle and healthy behaviors of older adults in Florianópolis, we obtained information on physical activity from older adults participating in "convivência" groups. Table 4 shows the average time spent in physical activity in leisure activities, transportation, and total physical activity in 2002, 2010. By 2010, the time spent in physical activity in transportation has increased significantly. These findings suggest that participating in "convivência" groups had a positive impact on physical activity lifestyle.

4. Discussion

In 2002, about 20% of older adults participated in "convivência" groups, whereas by 2010 the participation increased to 42%. By 2010, many more older adults were engaged in social activity with friends and many more were active in physical activity for leisure and transportation when compared to the 2002 survey. This study underscores the potential of "convivência" groups to promote socialization as well as healthy behaviors among the older adults population.

In Brazil, a number of public policy initiatives have facilitated the creation of community-based social groups. In 2003, the Brazilian National Public Policy for Older Adults [21] was implemented, and the Capital do Idoso—Idoso em Forma em Florianópolis program was started in 2006. The Capital do Idoso program was awarded a national recognition [35] for health and prevention services offered to the older adult population. Some researchers have documented the potential association of these public investments with reduced healthcare costs in the municipality. A study reported that selected hospital costs were reduced about 30% between 2006 and 2010 [36]. Other health indicators followed similar trends, such as improvements in sleeping quality, increased self-steem, and decreased usage of the public health system [37].

Table 1: Characteristics of the older adults living in Florianópolis, Brazil, in years 2002 and 2010.

	2002 Overall ($N = 875$)	2002 Male ($N = 437$)	2002 Female ($N = 438$)	2010 Overall ($N = 1705$)	2010 Male ($N = 616$)	2010 Female ($N = 1089$)
Age, years, mean (SD*)	71.5 (7.9)	71.4 (7.6)	71.7 (8.2)	70.7 (8.0)	70.6 (7.7)	70.9 (8.1)
Marital status, *N* (%)						
Married	536 (61.3)	363 (83.1)	173 (39.5)	993 (58.2)	505 (82)	488 (44.8)
Widow	251 (28.7)	43 (9.8)	208 (47.5)	481 (28.2)	41 (6.7)	440 (40.5)
Divorced	58 (6.6)	22 (5.0)	36 (8.2)	132 (7.7)	51 (8.3)	81 (7.4)
Single	30 (3.4)	9 (2.1)	21 (4.8)	99 (5.8)	19 (3.1)	80 (7.3)
Educational levels, *N* (%)						
None	175 (20.0)	80 (18.3)	95 (21.7)	161 (9.5)	50 (8.2)	111 (10.3)
1 to 8 years	476 (54.0)	215 (49.2)	261 (59.6)	767 (45.3)	256 (41.8)	511 (47.3)
More than 8 years	224 (25.6)	142 (32.5)	82 (18.7)	765 (45.2)	306 (50)	459 (42.5)
Socioeconomic levels (MMW**), *N* (%)						
Up to 2	398 (46.4)	142 (32.6)	256 (58.4)	1097 (64.3)	374 (60.7)	723 (66.4)
2 to 6	211 (24.6)	115 (26.4)	96 (21.9)	431 (25.3)	166 (26.9)	265 (24.3)
More than 6	248 (28.9)	167 (38.4)	81 (18.5)	177 (10.4)	76 (12.3)	101 (9.3)

* SD: Standard Deviation; ** MMW: Brazilian monthly minimum wage.

Table 2: Participation of the older adults in "convivência groups", 2002 and 2010 in Florianópolis, Brazil.

	Participate in "convivência" groups 2002 ($N = 875$)				Participate in "convivência" groups 2010 ($N = 1705$)			
	No	Yes	χ^2	*P* value	No	Yes	χ^2	*P* value
Gender *N* (%)								
Male	387 (55.1)	50 (29.1)	37524.0	<0.001	409 (41.4)	207 (28.8)	28.635	<0.001
Female	315 (44.9)	122 (70.9)			578 (58.6)	511 (71.2)		
Age *N* (%)								
60–69	323 (46)	79 (45.9)	1337.0	0.512	525 (53.2)	411 (57.2)	9600	0.008
70–79	264 (37.6)	59 (34.3)			323 (32.7)	241 (33.6)		
>80	115 (16.4)	34 (19.8)			139 (14.1)	66 (9.2)		
Marital status, *N* (%)								
Married	451 (64.2)	84 (48.8)	20.9	<0.001	592 (60)	401 (55.8)	7.267	0.064
Widow	23 (3.3)	7 (4.1)			60 (6.1)	39 (5.4)		
Divorced	50 (7.1)	8 (4.7)			81 (8.2)	51 (7.1)		
Single	178 (25.4)	73 (42.4)			254 (25.7)	227 (31.6)		
Educational levels, *N* (%)								
None	145 (20.7)	30 (17.4)	0.91	0.637	96 (9.8)	65 (9.1)	0.338	0.845
1 to 8 years	378 (53.8)	97 (56.4)			439 (44.8)	328 (45.9)		
More than 8 years	179 (25.5)	45 (26.2)			444 (45.4)	321 (45)		
Socio-economic levels (in MMW*), *N* (%)#								
Up to 2	317 (45.3)	80 (46.5)	0.72	0.868	643 (65.1)	454 (63.2)	0.777	0.678
2 to 6	168 (24)	43 (25)			242 (24.5)	189 (26.3)		
More than 6	201 (28.7)	47 (27.3)			102 (10.3)	75 (10.4)		
Physical activity level, *N* (%)								
Less active	501 (71.4)	118 (68.6)	0.51	0.266	530 (53.7)	330 (46)	9.953	0.002
More active	201 (28.6)	54 (31.4)			457 (46.3)	388 (54)		

* MMW: Brazilian monthly minimum wage; # 16 participants did not answer this question.

Table 3: Public programs available to older adults in Florianópolis, Brazil, between years 2002 and 2010.

Entities and programs	2002		2010	
	Number of groups	Number of older adults enrolled	Number of groups	Number of older adults enrolled
Municipality programs				
"Convivência"	93	3,500	105	4,509
Exercise and physical activity	57	1,670	111	4,311
Healthy lifestyle and disease management	2	50	94	1,250
University outreach programs				
Exercise and physical activity*	29	805	38	822
Senior education	22	596	45	997
Chambers of commerce (SESC) programs				
"Convivência" and senior social events	23	4,500	26	10,340
Exercise and physical activity	4	200	4	95
Senior education	1	20	13	374
Other programs				
Dancing schools for seniors	0	0	13	81
"Bailes" (dance clubs for seniors)	2	120	12	1,458

* Work Out Groups, Water Aerobics Groups, Swimming Groups, Dance Groups, Physical Activity to Parkinson Disease Groups, Sports Groups; Yoga Groups; Pilates Groups; Walking Groups; Bodybuilding Groups.

Table 4: Time spent in physical activity (leisure transportation and total) among older adults participating in "*convivência*" groups, in 2002 and 2010.

	2002 ($N = 875$)			2010 ($N = 1705$)			χ^2	P value
Participate in senior social groups, N (%)	172 (19.7)			718	42.1		128.65	<0.001
Visit relatives, N (%)	637 (72.8)			1194	71.2		0.77	N.S.
Visit friends	607 (69.4)			1256	74.9		8.76	0.003
Overall physical activity (IPAQ)								
Transportation PA, min/wk, mean (SE), 95% CI	48.3	8.1	32.3 to 64.2	120.8	4.7	1.5 to 130.1		<0.001
Transportation PA*, min/wk, mean (SE), 95% CI	46.7	7.7	31.5 to 61.9	125.8	5.5	5.1 to 136.4		<0.001
Leisure PA, mean (SE), 95% CI	108.1	7.1	94.1 to 122.1	128.7	5.2	8.5 to 138.8		0.004
Leisure PA*, mean (SE), 95% CI	113.0	7.8	97.8 to 128.2	130.2	5.5	9.5 to 141.0		0.015

*Adjusted by age, sex, BMI, education, and income.

Our findings suggest that older adults participating in "convivência" groups visited friends more often in 2010 than they did in 2002. In 2010, they were more engaged in other social groups available in the community. Also, in 2010 they spent more time in physical activity during leisure time and for transportation when compared to 2002. Our data suggest that participation in "convivência" groups may have played a role in facilitating positive health behaviors among group members.

A study conducted with older women in Canada examined the influence that social opportunities have on health and functioning capacity [38]. The results of this study showed that as the older women felt more accepted, they presented with less prevalence of diseases; in addition, they had enhanced functional capacity to perform household chores and other daily activities.

The demographic characteristics of the participants in his study are broadly consistent with profile of older Brazilians as a whole.

The aging process is often accompanied by declines in opportunities for social interactions and for establishing new relationships [40]. Retirement from working activities, children leaving home, and loss of loved ones are associated with increased social isolation among older adults [41]. The initial purpose of the Capital do Idoso program was not to promote social integration among the underserved older population of the city, but rather to develop educational activities that target disease prevention, health promotion, therapy, and rehabilitation. Shortly after its start, it became apparent that social engagement of older adults was one of the program's most successful outcomes. It was apparent how socially engaged participants became, and how much more frequently they would leave home to visit their friends. The programs also stimulated an increase in physical activity during transportation and leisure activities. Older adults are the most vulnerable age group for physical inactivity, and they are the least likely to meet physical activity guidelines [42].

The present study has certain limitations that need to be taken into account when considering the study and its contributions. The most important limitation lies in the fact that we did not include control groups, which would have assisted with the interpretation of findings, such as the participation in convivência groups and the positive impact of these groups on health behaviors. For example, it is not clear whether participants in convivência groups differed in their physical activity levels from the general population. In addition, our results may have been influenced by other aspects that were out of our study scope and control, such as increase of regional or national public health campaigns and resources.

In summary, our study suggests that the establishment of convivência groups made a difference to the lives of older adults in Florianópolis. Participating in these groups helped them to be socially engaged and to live actively. Most cities in Brazil have established some kind of convivência group. With this in mind, we can build a new culture of healthy and active aging throughout Brazil, as proposed by the WHO in 2002 [43].

Acknowledgments

This research was supported in part by the Brazilian Ministry of Health, Accord no. 4345/01; FAPEU Project 134-2001, and National Council for Scientific and Technological Development (CNPq), Procedure no. 520824/1997-0/SU and, Process no. 569834/2008 2, Public Notice no. 06/2008-B. Deep thanks are due to to all collaborators at IBGE and the Federal University of Santa Catarina.

References

[1] R. H. Binstock and L. K. Geoge, *Handbook of Aging and the Social Sciences*, Academic Press, 7th edition, 2010.

[2] S. A. Haslam, J. Jetten, T. Postmes, and C. Haslam, "Social identity, health and well-being: An emerging agenda for applied psychology," *Applied Psychology*, vol. 58, no. 1, pp. 1–23, 2009.

[3] L. Brown, M. Abigania, D. Warburton, and S. Brown, "Validation of QF-PCR for prenatal aneuploidy screening in the United States," *Prenatal Diagnosis*, vol. 26, no. 11, pp. 1068–1074, 2006.

[4] C. H. Cooley, *Social Organization: A Study of the Larger Mind*, Pan-American Copyright Conventions, New Brunswick, Canada, 4th edition, 1909.

[5] Y. L. Michael, L. F. Berkman, G. A. Colditz, and I. Kawachi, "Living arrangements, social integration, and change in functional health status," *American Journal of Epidemiology*, vol. 153, no. 2, pp. 123–131, 2001.

[6] M. V. Zunzunegui, F. Béland, and A. Otero, "Support from children, living arrangements, self-rated health and depressive symptoms of older people in Spain," *International Journal of Epidemiology*, vol. 30, no. 5, pp. 1090–1099, 2001.

[7] M. V. Zunzunegui, A. Koné, M. Johri, F. Béland, C. Wolfson, and H. Bergman, "Social networks and self-rated health in two French-speaking Canadian community dwelling populations over 65," *Social Science and Medicine*, vol. 58, no. 10, pp. 2069–2081, 2004.

[8] E. Saito, Y. Sagawa, and K. Kanagawa, "Social support as a predictor of health status among older adults living alone in Japan," *Nursing and Health Sciences*, vol. 7, no. 1, pp. 29–36, 2005.

[9] M. C. Resende, V. M. Bones, I. S. Souza, and N. K. Guimaraes, "Rede de relações sociais e satisfação com a vida de adultos e idosos," *Psicologia para a America Latina*, no. 5, 2006.

[10] Y. Koizumi, S. Awata, S. Kuriyama et al., "Association between social support and depression status in the elderly: Results of a 1-year community-based prospective cohort study in Japan," *Psychiatry and Clinical Neurosciences*, vol. 59, no. 5, pp. 563–569, 2005.

[11] S. Pin, E. Guilley, D. Spini, and C. Lalive D'Epinay, "The impact of social relationships on the maintenance of independence in advanced old age: Findings of a Swiss longitudinal study," *Zeitschrift fur Gerontologie und Geriatrie*, vol. 38, no. 3, pp. 203–209, 2005.

[12] S. C. J. Yeh and Y. Y. Liu, "Influence of social support on cognitive function in the elderly," *BMC Health Services Research*, vol. 3, article 1, pp. 1–9, 2003.

[13] M. V. Zunzunegui, B. E. Alvarado, T. Del Ser, and A. Otero, "Social networks, social integration, and social engagement determine cognitive decline in community-dwelling Spanish older adults," *Journals of Gerontology*, vol. 58, no. 2, pp. S93–S100, 2003.

[14] A. A. Eyler, R. C. Brownson, R. J. Donatelle, A. C. King, D. Brown, and J. F. Sallis, "Physical activity social support and middle- and older-aged minority women: results from a US survey," *Social Science and Medicine*, vol. 49, no. 6, pp. 781–789, 1999.

[15] W. K. Mummery, W. Lauder, G. Schofield, and C. Caperchione, "Associations between physical inactivity and a measure of social capital in a sample of Queensland adults," *Journal of Science and Medicine in Sport*, vol. 11, no. 3, pp. 308–315, 2008.

[16] F. A. Treiber, T. Baranowski, D. S. Braden, W. B. Strong, M. Levy, and W. Knox, "Social support for exercise: relationship to physical activity in young adults," *Preventive Medicine*, vol. 20, no. 6, pp. 737–750, 1991.

[17] R. K. Dishman, "Motivating older adults to exercise," *Southern Medical Journal*, vol. 87, no. 5, pp. S79–S82, 1994.

[18] R. P. Veras and S. Dutra, *Questionário BOAS*, Brazil Old Age Schedule, Versão, 2000-2001.

[19] G. Z. Mazo, M. A. Lopes, and T. R. B. Benedetti, *Atividade física e o idoso: Concepção Gerontológica*, Porto Alegre, Brazil, 3rd edition, 2009.

[20] T. R. B. Benedetti, Atividade física*: uma perspectiva de promoção da saúde do idoso no município de Florianópolis [Ph.D. thesis]*, Enfermagem, Universidade Federal de Santa Catarina, Florianópolis, Brazil, 2004.

[21] M. D. Saude, *Estatuto do Idoso*, no. 10.741, Senado Federal, Subsecretaria de Edições Técnicas, Brasília, Brazil, 2003.

[22] Organization U-UN, *Second World Assembly on Ageing*, Madrid, Spain, 2002.

[23] V. N. M. Gomes, *Cartilha: atuação da Ação Social Arquidiocesana na assessoria a dinamização de Grupos de Idosos*, Mimeo, Florianópolis, Santa Catarina, Brazil, 2001.

[24] M. Salgado, *Velhice: uma nova questão social*, vol. 2, SESC, São Paulo, Brazil, 1982.

[25] G. Z. Mazo, J. A. P. S. Mota, L. H. T. Gonçalves, and M. G. Matos, "Nível de atividade física, condições de saúde e características sócio-demográficas de mulheres idosas brasileiras," *Revista Portuguesa de Ciencia do Desporto*, vol. 5, no. 2, pp. 202–212, 2005.

[26] P. L. C. Borges, R. P. Bretas, S. F. Azevedo, and J. M. M. Barbosa, "Perfi l dos idosos frequentadores de grupos de convivência em Belo Horizonte, Minas Gerais, Brasil," *Cadernos de Saúde Pública*, vol. 24, no. 12, pp. 2798–2809, 2008.

[27] G. Z. Mazo, J. A. P. S. Mota, L. H. T. Gonçalves, M. G. Matos, and J. Carvalho, "Actividade física e qualidade de vida de mulheres idosas da cidade de Florianópolis, Brasil," *Revista Portuguesa de Ciência do Desporte*, vol. 8, no. 3, p. 9, 2008.

[28] T. R. B. Benedetti, É. L. Petroski, and L. H. T. Goncalves, *Perfil dos idosos do Município de Florianópolis*, Palotti, Florianópolis, Brazil, 2004.

[29] E. d'Orsi, *Condições de saúde da população idosa do município de Florianópolis, Santa Catarina: estudo de base populacional*, Conselho Nacional de Desenvolvimento Científico e Tecnológico, Florianopolis, Brazil, 2008.

[30] IBGE, "Estimativas populacionais para o TCU," Estimativas da população para 1° de Julho de 2009, Rio de Janeiro, Brazil, 2009.

[31] C. L. Craig, A. L. Marshall, M. Sjöström et al., "International physical activity questionnaire: 12-country reliability and validity," *Medicine and Science in Sports and Exercise*, vol. 35, no. 8, pp. 1381–1395, 2003.

[32] T. R. B. Benedetti, G. Z. Mazo, and M. V. G. Barros, "Aplicação do Questionário Internacional de Atividades Físicas para avaliação do nível de atividades físicas de mulheres idosas: validade concorrente e reprodutibilidade teste-reteste," *Revista Brasileira de Ciência e Movimento*, vol. 12, no. 1, pp. 25–35, 2004.

[33] T. R. B. Benedetti, P. D. C. Antunes, C. R. Rodriguez-Añez, G. Z. Mazo, and É. L. Petroski, "Reprodutibilidade e validade do Questionário Internacional de Atividade Física (IPAQ) em homens idosos," *Revista Brasileira de Medicina do Esporte*, vol. 13, no. 1, pp. 17–28, 2007.

[34] T. R. B. Benedetti, L. H. T. Gonçalves, and J. A. P. S. Mota, "Uma proposta de política pública de atividade física para idosos," *Texto Contexto em Enfermagem*, vol. 16, no. 3, p. 12, 2007.

[35] E. Abril, Premio Saúde, 2010, http://saude.abril.com.br/premiosaude/2010/finalistas-prevencao.shtml.

[36] M. Castanhel, N. F. Oliveira, and E. Gonçalves, *Capital idoso*, Secretaria da Saúde de Florianópolis, Florianópolis, Brazil, 2011.

[37] S. T. Meurer, T. R. B. Benedetti, and G. Z. Mazo, "Teoria da autodeterminação: compreensão dos fatores motivacionais e autoestima de idosos praticantes de exercícios físicos," *Revista Brasileira de Atividade Física & Saúde*, vol. 2011, 2011.

[38] K. A. Greiner, C. Li, I. Kawachi, D. C. Hunt, and J. S. Ahluwalia, "The relationships of social participation and community ratings to health and health behaviors in areas with high and low population density," *Social Science and Medicine*, vol. 59, no. 11, pp. 2303–2312, 2004.

[39] IBGE, *Síntese de Indicadores Sociais: uma análise das condições de vida da população brasileira*, Rio de Janeiro, Brazil, 2010.

[40] C. E. Sluzki, *A Rede Social na Prática Sistêmica*, Psicólogo Cd, São Paulo, Brazil, 1997.

[41] M. A. R. C. Domingues, *Mapa Mínimo das relações: adaptação de um instrumento gráfico para configuração de rede de apoio social do idoso [Ph.D. thesis]*, Faculdade de Saúde Pública, Universidade de São Paulo, São Paulo, Brazil, 2000.

[42] USDHHS, *Physical activity guidelines for Americans*, Washington, DC, USA, 2008.

[43] WHO, *Active Aging: A Polyce Framework*, Course AaL, WHO, Geneva, Switzerland, 2002.

Hindawi Publishing Corporation
Journal of Aging Research
Volume 2012, Article ID 243958, 9 pages
doi:10.1155/2012/243958

Review Article

Does Physical Activity Increase Life Expectancy? A Review of the Literature

C. D. Reimers,[1] G. Knapp,[2] and A. K. Reimers[3]

[1] *Klinik für Neurologie, Zentralklinik Bad Berka, Robert-Koch-Allee 9, 99438 Bad Berka, Germany*
[2] *Fakultät Statistik, Technische Universität Dortmund, 44221 Dortmund, Germany*
[3] *Sportwissenschaft, Universität Konstanz, Universitätsstraße 10, 78457 Konstanz, Germany*

Correspondence should be addressed to C. D. Reimers, c.d.reimers@t-online.de

Received 18 February 2012; Revised 19 March 2012; Accepted 13 April 2012

Academic Editor: Iris Reuter

Physical activity reduces many major mortality risk factors including arterial hypertension, diabetes mellitus type 2, dyslipidemia, coronary heart disease, stroke, and cancer. All-cause mortality is decreased by about 30% to 35% in physically active as compared to inactive subjects. The purpose of this paper was to synthesize the literature on life expectancy in relation to physical activity. A systematic PubMed search on life expectancy in physically active and inactive individuals was performed. In addition, articles comparing life expectancy of athletes compared to that of nonathletes were reviewed. Results of 13 studies describing eight different cohorts suggest that regular physical activity is associated with an increase of life expectancy by 0.4 to 6.9 years. Eleven studies included confounding risk factors for mortality and revealed an increase in life expectancy by 0.4 to 4.2 years with regular physical activity. Eleven case control studies on life expectancy in former athletes revealed consistently greater life expectancy in aerobic endurance athletes but inconsistent results for other athletes. None of these studies considered confounding risk factors for mortality. In conclusion, while regular physical activity increases life expectancy, it remains unclear if high-intensity sports activities further increase life expectancy.

1. Introduction

The most important causes of death in Western industrialized countries are cardio- and cerebrovascular diseases and malignancies. For instance, in Germany in 2008, 68.6% of all women and 65.9% of all men died from these diseases. In contrast, the third most frequent cause of death are respiratory diseases which cause less than 10% of deaths each year (Table 1). Important risk factors for cardio- and cerebrovascular diseases include smoking, arterial hypertension, obesity, diabetes mellitus, and dyslipidemia along with atrial fibrillation for ischemic strokes [1, 2].

Regular physical activity reduces the risk of and/or improves many diseases and conditions including arterial hypertension, diabetes mellitus type 2, dyslipidemia, obesity, coronary heart disease, chronic heart failure [3, 4], and chronic obstructive pulmonary disease [4]. In addition, the risk of colon [3, 5], breast [4, 5], and possibly endometrial, lung, and pancreatic cancer is reduced [5] (Table 2).

The relative risk of death is approximately 20% to 35% lower in physically active and fit persons compared to that in inactive and unfit persons [6, 7]. Physical inactivity represents a major independent risk factor for mortality accounting for up to 10% of all deaths in the European region [3]. Hence, because a 40% lower mortality rate corresponds to an approximately 5-year higher life expectancy [8], one would expect an approximately 3.5- to 4.0-year higher life expectancy in physically active persons compared to that in inactive persons.

The purpose of this review was to synthesize the literature on life expectancy in relation to physical activity. Specifically, cohort studies on physically active and inactive subjects were reviewed to detect a possible difference in life expectancy between these subject groups. In addition, cohort studies on athletes and non-athletes were reviewed to detect a possible difference in life expectancy between these subject groups.

Table 1: Number of deaths (percentage of total number of deaths) for the three most frequent causes of death for women and men in Germany in 2008 [9].

	Women (per 100,000 persons/y)	Men (per 100,000 persons/y)
Total	467.3 (100.0%)	720.5 (100.0%)
Vascular diseases	186.4 (39.9%)	263.6 (36.6%)
Coronary heart disease	61.8 (13.2%)	117.4 (16.3%)
Strokes	35.8 (7.7%)	41.6 (5.8%)
Malignant tumors	134.0 (28.7%)	210.9 (29.3%)
Respiratory diseases	27.7 (5.9%)	53.3 (7.4%)

2. Method

To identify all relevant articles about cohort studies investigating the life expectancy of physically active versus inactive persons, a systematic literature search was conducted in the electronic bibliographical database PubMed (http://www.ncbi.nlm.nih.gov/pubmed/). We only searched for English-language peer-reviewed journal articles using the search terms "(life expectancy OR longevity) AND (physical activity OR exercise OR sport)" (last update January 3, 2012). A total of 1,932 articles were found. Using the abstracts and/or titles, forty-one of these articles were identified as cohort studies comparing mortality and/or life expectancy of physically active and inactive persons. However, of these, only 13 articles presented detailed data on life expectancy for both groups.

Subsequently, a search was performed using the terms "(life expectancy OR longevity) AND athlete" to find articles on life expectancy in (former) athletes. Sixty-six articles were found. Additional publications were identified using the search term "(life expectancy OR longevity) AND (physical activity OR exercise OR sport)." Of all these articles, 21 articles investigated mortality and/or life expectancy of (former) athletes. Eleven of these articles presented detailed data on life expectancy of athletes compared to that of a control group.

The remaining life expectancy for physically active and inactive individuals or the difference in remaining life expectancy between the two groups, respectively, were reported in the articles. However, the attained ages of subject groups differed between studies. Because the differences of remaining life expectancies cannot be assumed to be independent from the attained age, we report the results stratified for attained age. Within each stratum of attained age, the results were presented for both sexes.

Only five articles [16–20] reported confidence intervals for the difference in remaining life expectancies between groups. In four articles [16, 18–20], a parametric bootstrap procedure was used, and in one publication [17] a nonparametric maximum likelihood approach was used. Consequently, we did not combine the results in a meta-analysis model.

Because a meta-analysis model was not appropriate, all data found in the literature search were reported despite some overlaps between the cohort studies. For instance, Jonker et al. [18] and Nusselder et al. [19] used data from the Framingham Heart Study and Paffenbarger et al. [21–24] used data from the Harvard Alumni cohort. While most studies reported results based on multivariate life-table analysis, the studies by Menotti et al. [25] and Pekkanen et al. [26] only reported the results of gained life expectancies for the cohorts based on classical survival analysis. Additionally, the study of Pekkanen et al. [26] reported the survival rate of men aged 40 to 65 years in the following 20 years and not the total life expectancy.

3. Results

Thirteen cohort studies presented data on life expectancy in physically active individuals compared to that in physically inactive control subjects (Table 3). All studies reported a higher life expectancy in physically active subjects, ranging from 0.43 to 6.9 additional years (mean ± one standard deviation, men: 2.9 ± 1.3 years, women: 3.9 ± 1.8 years). Eleven studies considered confounding factors that could affect life expectancy, such as body mass index, blood pressure, diabetes mellitus, dyslipidemia, cardiovascular and lung diseases, cancer, smoking, or alcohol consumption [16–25, 27]. The additional life expectancy in physically active compared to inactive persons in these studies ranged between 0.43 and 4.21 years (2.7 ± 1.1 years). The physically most active groups, included in the estimations of life expectancy, participated in moderate to high leisure time [16, 27–29] or leisure time and all-day activities [17–25].

The median increase of life expectancy of men and women in the eight studies presenting data on both sexes amounted to 3.7 years each. Physical activity during leisure time seems to increase life expectancy more effectively than total physical activity (all-day, professional, or leisure time activity altogether; professional physical activity alone has not been studied): 3.4 added years due to total activities and 4.7 added years (median values) due to leisure time activities in women, 1.9 and 3.9 added years, respectively, in men. The number of studies, however, is too low for a statistical analysis. Furthermore, the description of the amount of physical activity in the active and inactive groups are too heterogeneous for any statistical correlation between the amount of activity and the added years of life.

The eleven case control studies on life expectancy of athletes, mostly elite athletes (Table 4), reported a mean life expectancy that was between 5.0 years lower and 8.0 years higher than that of the nonathlete control groups. Aerobic endurance sports resulted on average in a 4.3 to 8.0 years higher life expectancy and team sports activities on average in a 5.0 years lower to about 5 years higher life expectancy compared to that for normal physical activity. Only one study presented data on strength sports and reported a slightly higher life expectancy compared to that for normal physical activity. None of these studies considered any confounding factor that could affect life expectancy.

Table 2: Preventive effects of regular physical activity on major risk factors for cardio- and cerebrovascular diseases and cancer.

Author(s)	Risk factor	Effect of regular physical activity on the risk factor in healthy subjects
Adami et al. [3], Halle and Schoenberg [10], Warburton et al. [6], Warburton et al. [11]	Colon cancer	Incidence −30% to −40%
Adami et al. [3], Monninkhof et al. [12], Warburton et al. [6]	Breast cancer	Incidence −20% to −50%
Walker et al. [13], Warburton et al. [11]	Type 2 diabetes mellitus	Incidence −28% to −59%
G. A. Kelley and K. S. Kelley [14]	Dyslipidemia	HDL cholesterol +11%
Pedersen and Saltin [4]	Arterial hypertension	Systolic and diastolic blood pressure −3.84/−2.58 mmHg
Pedersen and Saltin [4]	Obesity	Increased chance to maintain body weight
Warburton et al. [11], Reimers et al. [15]	Stroke	Incidence −27% to −40%

4. Discussion

The purpose of this review was to synthesize the literature on life expectancy in relation to physical activity. Being physically active indeed appears to be associated with a higher life expectancy. Samitz et al. [7] as well as Warburton et al. [11] reported a mean reduction of mortality of 31% to 35% in persons who participate in regular leisure-time or daily life physical activity compared to that in inactive persons. Assuming a 40% lower mortality corresponding to a 5-year higher life expectancy [8], regular physical activity should increase mean life expectancy by approximately (31% to 35%)/40% × 5 years or by 3.9 to 4.4 years. Indeed, the few articles that presented data on life expectancy in physically active individuals reported a 0.43- to 6.9-year higher life expectancy. High-quality studies considering confounding factors that could affect mortality reported a 0.43- to 4.21-years higher life expectancy in physically active compared to inactive persons. The wide range of years added cannot be explained based on the published data.

The studies that standardized extended life estimates for confounding factors [16, 18–25, 27] virtually calculated a net gain in life expectancy by being physically active. However, the actual increase in life expectancy should be much higher because of favorable effects of physical activity on other risk factors for mortality such as arterial hypertension, glucose and lipid metabolism, coronary heart disease, stroke, or malignancies (Table 1). In fact, nonsmoking, normal weight, and physically fit men live on average 12 years (95% confidence interval, 8.6 to 14.6 years) longer than smoking, overweight, and physically unfit control subjects [41]. Subjects who never smoked, follow a healthy diet, are adequately physically active, and consume only moderate alcohol have a mean life expectancy that is 11.1 years longer than those who practice none of these healthy life behaviors [42].

The mechanisms underlying the net effect of physical activity are speculative and include reduction of triglyceride and apolipoprotein B concentrations, increase of high-density lipoproteins and tissue plasminogen activator activity, and reduction of coronary artery calcium resulting in reduced risks of vascular diseases, which carry the strongest mortality risk [43]. In addition, regular physical activity increases the endurance of cells and tissues to oxidative stress, vascularization, and energy metabolism [44].

According to the results of the meta-analysis on all-cause mortality in relation to physical activity performed by Samitz et al. [7], vigorous physical activity (>6 metabolic equivalents (MET)) reduces mortality slightly, but this reduction is significantly more pronounced than that for moderate activity (3–6 MET).

A greater life expectancy is not associated with more years of being frail and depending on assistance. In contrary, Nusselder et al. [20] reported a gain of disability-free years of life with a higher life expectancy.

The few data available on life expectancy in athletes who were much more physically active than the average individual are inconclusive. All studies proved an increased life expectancy in endurance athletes ranging between 2.8 to 8.0 added years. This gain is probably higher than that found for persons performing vigorous physical activity in the cohort studies. In team sports and other sports disciplines, life expectancy may fall below or be above that of the control groups. However, data on health behaviors of these athletes other than their physical activity during their active sports career such as smoking, food, and alcohol consumption are not available. Thus, the effect of elite sports activities on life expectancy warrants further investigation.

In summary, as expected from numerous prospective cohort studies on all-cause mortality in physically active and physically inactive persons, estimates on life expectancy in relation to physical activity indicate additional years of life in active subjects: the conservative estimate of the net increase in life expectancy with physical activity is about 2–4 years but presumably even greater because of the positive influence of physical activity on major risk factors for mortality.

Table 3: Cohort studies comparing the life expectancy of physically active and inactive persons.

Sex	Age (class) at start of followup	Authors	Country	Number of individuals, duration of follow-up	Estimate additional years of life (95% CI) (years)	Activity of the "active" group	Activity of the "inactive" group	Confounding factors
Women	30	Fraser and Shavlik [17]	USA	#12 y	2.19 (0.92–3.45)*	At least 3 times per week vigorous all-day or sports activities ≥ 15 min.	Less than 3 times per week intensive all-day and sportive activities ≥15 min.	Vegetarian/nonvegetarian, high/low nut consumption, body mass index, never/past smoker, hormone replacement therapy
	30	Wen et al. [27]	Taiwan	216.910 8.05 ± 4.02 y	3.67 84.08 versus 87.75	Very vigorous physical activity (≥25.5 MET·h/week) during leisure time	Inactivity (<3.75 MET·h/week) during leisure time	Age, sex, education, physical work, smoking, alcohol consumption, diabetes mellitus, arterial hypertension, cancer, fasting glucose, systolic blood pressure, total cholesterol, body mass index
	45	Bélanger et al. [28]	Canada	#2 y	6.9 81.7 versus 88.6	≥1.5 kcal/kg/d energy expenditure during leisure time	<1.5 kcal/kg/d energy expenditure during leisure time	
	50	Jonker et al. [18]	USA (Framingham Heart Study)	2.813 12 y	3.7 (2.6–4.9) 82.3 versus 86.0	High physical activity level (>33 METs/d)	Low physical activity level (<30 METs/d)	Age, education, smoking, marital status, cardiovascular and lung diseases, cancer, left ventricular hypertrophy, arthritis, ankle edema, total cholesterol, familial history of diabetes mellitus
	50	Nusselder et al. [20]	The Netherlands	1.447 2 y	1.8 (0.5–2.7) 76.5 versus 78.3	Walking, biking, gardening, sports >17.33 METs/week	Walking, biking, gardening, sports <12 METs/week	Age, sex, education, cardiovascular disease, cancer, COPD, arthritis, back complaints, neurological diseases

Table 3: Continued.

Sex	Age (class) at start of followup	Authors	Country	Number of individuals, duration of follow-up	Estimate additional years of life (95% CI) (years)	Activity of the "active" group	Activity of the "inactive" group	Confounding factors
	50	Nusselder et al. [19]	USA (Framingham Heart Study)	2.873 12 y	3.4 (2.3–4.5) 82.7 versus 86.1	High physical activity level (>33 METs/d)	Low physical activity level (<30 METs/d)	Age, sex, education, marital status, smoking, body mass index, blood pressure, cancer, diabetes mellitus, left ventricular hypertrophy, ankle edema, any pulmonary disease, smoking
	65	Ferrucci et al. [29]	USA	5.215 6 y	Nonsmoker: 5.7 77.7 versus 83.4 Smoker: 4.2 76.1 versus 80.3	High physical activity (gardening, walking, vigorous exercise) each once per week or several times per month *or* one of these activities several times per week and another activity once per week or several times per month	Activities as in the active group at most once per month	
Men	30	Fraser and Shavlik [17]	USA	#12 y	2.1 (0.4–3.9)*	At least 3 times per week vigorous all-day or sports activities ≥15 min.	Less than 3 times per week intense all-day and sportive activities ≥15 min.	Vegetarian/nonvegetarian, high/low nut consumption, body mass index, never/past smoker, hormone replacement therapy
	30	Wen et al. [27]	Taiwan	199.265 8.05 ± 4.02 y	4.21 80.37 versus 84.58	Very vigorous physical activities (≥25.5 MET·h/week) during leisure time	Inactivity (<3.75 MET·h/week) during leisure time	Age, sex, education, physical work, smoking, drinking, diabetes mellitus, arterial hypertension, history of cancer, fasting blood glucose, systolic blood pressure, total cholesterol, body mass index
	35–39	Paffenbarger et al. [21]	USA (Harvard Alumni)	16.936 16 y	1.5*	Physical activities (walking, climbing stairs, sports) ≥2.000 kcal/week	Physical activities (walking, climbing stairs, sports) <2.000 kcal/week	Age, cigarette smoking, arterial hypertension, body mass index, age of parental death

Table 3: Continued.

Sex	Age (class) at start of followup	Authors	Country	Number of individuals, duration of follow-up	Estimate additional years of life (95% CI) (years)	Activity of the "active" group	Activity of the "inactive" group	Confounding factors
	35–39	Paffenbarger et al. [22]	USA (Harvard Alumni)	16.936 12–16 y	2.51*	Physical activities (walking, climbing stairs, sports) ≥2.000 kcal/week	Physical activities (walking, climbing stairs, sports) <500 kcal/week	Age, cigarette smoking, arterial hypertension, body mass index, age of parental death
	40–59	Menotti et al. [25]	Italy	1.712 40 y	1.6*	Physically active	Sedentary	Age, family history of both parents, mean blood pressure, serum cholesterol, mid-arm circumference, forced exspiratory volume, chronic diseases (cardiovascular, diabetes mellitus, cancer), corneal arcus, xanthelasma, body mass index
	45	Bélanger et al. [28]	Canada	#2 y	3.9 76.9 versus 80.8	≥1.5 kcal/kg/d energy expenditure during leisure time	<1.5 kcal/kg/d energy expenditure during leisure time	
	45–54	Paffenbarger et al. [23]	USA (Harvard Alumni)	10.269 8 y	0.43*	Physical activities ≥2.000 kcal/week	Physical activities <2.000 kcal/week	Age, cigarette smoking, arterial hypertension, overweight, early parental death
	45–54	Paffenbarger et al. [24]	USA (Harvard Alumni)	14.785 11 y	1.78*	Physical activity (walking, stair climbing, sports, or recreational activities) increased from <1.500 to ≥1.500 kcal/week	Physical activity (walking, stair climbing, sports, or recreational activities) continuing <1.500 kcal/week	Age, cigarette smoking, arterial hypertension, overweight, alcohol consumption, early parental death, chronic diseases
	50	Jonker et al. [18]	USA (Framingham Heart Study)	2.396 12 y	4.1 (2.8–5.4) 75.3 versus 79.4	High physical activity level (>30 METs/d)	Low physical activity level (<30 METs/d)	Age, education, smoking, marital status, cardiovascular and lung diseases, cancer, left ventricular hypertrophy, arthritis, ankle edema, total cholesterol, familial history of diabetes mellitus

Table 3: Continued.

Sex	Age (class) at start of followup	Authors	Country	Number of individuals, duration of follow-up	Estimate additional years of life (95% CI) (years)	Activity of the "active" group	Activity of the "inactive" group	Confounding factors
	50	Nusselder et al. [20]	The Netherlands	1.519 2 y	2.9 (0.9–4.3) 74.8 versus 76.7	Walking, biking, gardening, sports >17.33 METs/week	Walking, biking, gardening, sports <12 METs/week	Age, sex, education, cardiovascular disease, cancer, chronic obstructive pulmonary disease, arthritis, back complaints, neurological diseases
	50	Nusselder et al. [19]	USA (Framingham Heart Study)	2.336 12 y	3.5 (2.5–4.6) 76.4 versus 80.0	High physical activity level (>33 METs/d)	Low physical activity level (<30 METs/d)	Age, sex, education, marital status, body mass index, blood pressure, cancer, diabetes mellitus, left ventricular hypertrophy, ankle edema, any pulmonary disease, smoking
	50	Byberg et al. [16]	Sweden	2.205 35 y	2.3 (1.3–3.3)*	Regularly hard physical training or competitive sport or any active recreational sports or heavy gardening at least 3 hours every week	Spending most of the time reading, watching TV, going to the cinema, or engaging in other, mostly sedentary activities	Smoking, weight and height, alcohol use, obesity, diabetes mellitus, musculoskeletal, neurological, or psychiatric disorders, blood pressure, antihypertensive drugs, total serum cholesterol, educational level, socioeconomic group
	65	Ferrucci et al. [29]	USA	3.389 6 y	Nonsmoker: 5.2 76.0 versus 81.2 Smoker: 3.4 74.5 versus 77.9	High physical activity (gardening, walking, vigorous exercise) each once per week or several times per month *or* one of these activities several times per week and another activity once per week or several times per month	Activities as in the active group maximally once per month	

* Total life expectancy not presented, # number of individuals not differentiated between men and women.

Table 4: Case control studies presenting life expectancy of (former) athletes compared to that of control subjects.

Author(s)	Type of sports	Reduction/increase in life expectancy (y)
Prout [30]	Endurance sports (college rowers from Harvard and Yale)	+6.3
Sarna et al. [31]	Endurance sports (long distance running, cross-country skiing)	+5.7
Karvonen [32], Karvonen et al. [33]	Endurance sports (cross-country skiing)	+2.8 to +4.3
Sanchis-Gomar et al. [34]	Endurance sports (Tour de France cyclists)	+8.0
Sarna et al. [31], Sarna and Kaprio [35]	Power sports (throwing sports, wrestling, weight lifting, boxing)	+1.6
Sarna et al. [31], Sarna and Kaprio [35]	Team sports (ice hockey, soccer, basketball, other outdoor sports)	+4.0
Abel and Kruger [36]	Team sports (baseball)	−5.0
Abel and Kruger [37]	Team sports (baseball)	+4 to 5
Kuss et al. [38]	Team sports (German international soccer players)	−1.9 J. (+0.6 to −3.2)
Hudec et al. [39]	Various sports disciplines	−0.38
Rook [40]	Various sports disciplines	+1.03

Abbreviations

CI: Confidence interval
COPD: Chronic obstructive pulmanry disease
d: Day
h: Hour(s)
MET: Metabolic equivalent value (1 MET = 1 kcal per h per kg of bodyweight)
Min.: Minute(s)
TV: Television
vs: Versus
y: Year(s).

References

[1] S. E. Straus, S. R. Majumdar, and F. A. McAlister, "New evidence for stroke prevention: scientific review," *Journal of the American Medical Association*, vol. 288, no. 11, pp. 1388–1395, 2002.

[2] S. Ebrahim, F. Taylor, K. Ward, A. Beswick, M. Burke, and G. Davey Smith, "Multiple risk factor interventions for primary prevention of coronary heart disease," *Cochrane Database of Systematic Reviews*, vol. 1, Article ID CD001561, 2011.

[3] P. E. Adami, A. Negro, N. Lala, and P. Martelletti, "The role of physical activity in the prevention and treatment of chronic diseases," *Clinica Terapeutica*, vol. 161, no. 6, pp. 537–541, 2010.

[4] B. K. Pedersen and B. Saltin, "Evidence for prescribing exercise as therapy in chronic disease," *Scandinavian Journal of Medicine and Science in Sports*, vol. 16, no. 1, pp. 3–63, 2006.

[5] C. M. Ulrich, J. Wiskemann, and K. Steindorf, "Physiologic and molecular mechanisms linking physical activity to cancer risk and progression," *Bundesgesundheitsblatt Gesundheitsforschung Gesundheitsschutz*, vol. 55, no. 1, pp. 3–9, 2012.

[6] D. E. R. Warburton, C. W. Nicol, and S. S. D. Bredin, "Health benefits of physical activity: the evidence," *CMAJ*, vol. 174, no. 6, pp. 801–809, 2006.

[7] G. Samitz, M. Egger, and M. Zwahlen, "Domains of physical activity and all-cause mortality: systematic review and dose-response meta-analysis of cohort studies," *International Journal of Epidemiology*, vol. 40, no. 5, Article ID dyr112, pp. 1382–1400, 2011.

[8] B. Farahmand, G. Broman, U. De Faire, D. Vågerö, and A. Ahlbom, "Golf: a game of life and death—reduced mortality in Swedish golf players," *Scandinavian Journal of Medicine and Science in Sports*, vol. 19, no. 3, pp. 419–424, 2009.

[9] E. Gaber and M. Wildner, "Heft 52—Sterblichkeit, Todesursachen und regionale Unterschiede," in *Gesundheitsberichterstattung des Bundes*, E. Gaber and T. Ziese, Eds., Robert Koch-Institut, Berlin, Germany, 2011.

[10] M. Halle and M. H. Schoenberg, "Physical activity in the prevention and treatment of colorectal carcinoma," *Deutsches Ärzteblatt*, vol. 106, no. 44, pp. 722–727, 2009.

[11] D. E. R. Warburton, S. Charlesworth, A. Ivey, L. Nettlefold, and S. S. D. Bredin, "A systematic review of the evidence for Canada's Physical Activity Guidelines for Adults," *International Journal of Behavioral Nutrition and Physical Activity*, vol. 7, article 39, 2010.

[12] E. M. Monninkhof, S. G. Elias, F. A. Vlems et al., "Physical activity and breast cancer: a systematic review," *Epidemiology*, vol. 18, no. 1, pp. 137–157, 2007.

[13] K. Z. Walker, K. O'Dea, M. Gomez, S. Girgis, and R. Colagiuri, "Diet and exercise in the prevention of diabetes," *Journal of Human Nutrition and Dietetics*, vol. 23, no. 4, pp. 344–352, 2010.

[14] G. A. Kelley and K. S. Kelley, "Aerobic exercise and HDL2-C: a meta-analysis of randomized controlled trials," *Atherosclerosis*, vol. 184, no. 1, pp. 207–215, 2006.

[15] C. D. Reimers, G. Knapp, A. K. Reimers, and B. Griewing, "Physical activity and stroke," *Aktuelle Neurologie*. In press.

[16] L. Byberg, H. Melhus, R. Gedeborg et al., "Total mortality after changes in leisure time physical activity in 50 year old men: 35 Year follow-up of population based cohort," *British Journal of Sports Medicine*, vol. 43, no. 7, artice 482, 2009.

[17] G. E. Fraser and D. J. Shavlik, "Ten years of life is it a matter of choice?" *Archives of Internal Medicine*, vol. 161, no. 13, pp. 1645–1652, 2001.

[18] J. T. Jonker, C. De Laet, O. H. Franco, A. Peeters, J. Mackenbach, and W. J. Nusselder, "Physical activity and life expectancy with and without diabetes: life table analysis of the Framingham Heart Study," *Diabetes Care*, vol. 29, no. 1, pp. 38–43, 2006.

[19] W. J. Nusselder, O. H. Franco, A. Peeters, and J. P. MacKenbach, "Living healthier for longer: comparative effects of three heart-healthy behaviors on life expectancy with and without cardiovascular disease," *BMC Public Health*, vol. 9, article 487, 2009.

[20] W. J. Nusselder, C. W. N. Looman, O. H. Franco, A. Peeters, A. S. Slingerland, and J. P. Mackenbach, "The relation between non-occupational physical activity and years lived with and without disability," *Journal of Epidemiology and Community Health*, vol. 62, no. 9, pp. 823–828, 2008.

[21] R. S. Paffenbarger, R. T. Hyde, C. C. Hsieh, and A. L. Wing, "Physical activity, other life-style patterns, cardiovascular disease and longevity," *Acta Medica Scandinavica*, vol. 220, no. 711, pp. 85–91, 1986.

[22] R. S. Paffenbarger, R. T. Hyde, A. L. Wing, and C. C. Hsieh, "Physical activity, all-cause mortality, and longevity of college alumni," *The New England Journal of Medicine*, vol. 314, no. 10, pp. 605–613, 1986.

[23] R. S. Paffenbarger, R. T. Hyde, A. L. Wing, I. M. Lee, D. L. Jung, and J. B. Kampert, "The association of changes in physical-activity level and other lifestyle characteristics with mortality among men," *The New England Journal of Medicine*, vol. 328, no. 8, pp. 538–545, 1993.

[24] R. S. Paffenbarger, J. B. Kampert, I. M. Lee, R. T. Hyde, R. W. Leung, and A. L. Wing, "Changes in physical activity and other lifeway patterns influencing longevity," *Medicine and Science in Sports and Exercise*, vol. 26, no. 7, pp. 857–865, 1994.

[25] A. Menotti, M. Lanti, G. Maiani, and D. Kromhout, "Determinants of longevity and all-cause mortality among middle-aged men. Role of 48 personal characteristics in a 40-year follow-up of Italian Rural Areas in the Seven Countries Study," *Aging*, vol. 18, no. 5, pp. 394–406, 2006.

[26] J. Pekkanen, B. Marti, and A. Nissinen, "Reduction of premature mortality by high physical activity: a 20-year follow-up of middle-aged Finnish men," *The Lancet*, vol. 1, no. 8548, pp. 1473–1477, 1987.

[27] C. P. Wen, J. P. M. Wai, M. K. Tsai et al., "Minimum amount of physical activity for reduced mortality and extended life expectancy: a prospective cohort study," *The Lancet*, vol. 378, no. 9798, pp. 1244–1253, 2011.

[28] A. Bélanger, L. Martel, J. M. Berthelot, and R. Wilkins, "Gender differences in disability-free life expectancy for selected risk factors and chronic conditions in Canada," *Journal of Women and Aging*, vol. 14, no. 1-2, pp. 61–83, 2002.

[29] L. Ferrucci, G. Izmirlian, S. Leveille et al., "Smoking, physical activity, and active life expectancy," *American Journal of Epidemiology*, vol. 149, no. 7, pp. 645–653, 1999.

[30] C. Prout, "Life expectancy of college oarsmen," *JAMA*, vol. 220, no. 13, pp. 1709–1711, 1972.

[31] S. Sarna, T. Sahi, M. Koskenvuo, and J. Kaprio, "Increased life expectancy of world class male athletes," *Medicine and Science in Sports and Exercise*, vol. 25, no. 2, pp. 237–244, 1993.

[32] M. J. Karvonen, "Endurance sports, longevity, and health," *Annals of the New York Academy of Sciences*, vol. 301, pp. 653–655, 1977.

[33] M. J. Karvonen, H. Klemola, J. Virkajarvi, and A. Kekkonen, "Longevity of endurance skiers," *Medicine and Science in Sports and Exercise*, vol. 6, no. 1, pp. 49–51, 1974.

[34] F. Sanchis-Gomar, G. Olaso-Gonzalez, D. Corella, M. C. Gomez-Cabrera, and J. Vina, "Increased average longevity among the Tour de France cyclists," *International Journal of Sports Medicine*, vol. 32, no. 8, pp. 644–647, 2011.

[35] S. Sarna and J. Kaprio, "Life expectancy of former elite athletes," *Sports Medicine*, vol. 17, no. 3, pp. 149–151, 1994.

[36] E. L. Abel and M. L. Kruger, "The longevity of baseball hall of famers compared to other players," *Death Studies*, vol. 29, no. 10, pp. 959–963, 2005.

[37] E. L. Abel and M. L. Kruger, "Longevity of major league baseball players," *Research in Sports Medicine*, vol. 13, no. 1, pp. 1–5, 2005.

[38] O. Kuss, A. Kluttig, and K. H. Greiser, "Longevity of soccer players: an investigation of all German internationals from 1908 to 2006," *Scandinavian Journal of Medicine and Science in Sports*, vol. 21, no. 6, pp. e260–e265, 2011.

[39] A. Hudec, E. Mahoney, H. J. Montoye, H. Olson, and W. D. van Huss, "Study of the longevity and morbidity of college athletes," *JAMA*, vol. 162, no. 12, pp. 1132–1134, 1956.

[40] A. ROOK, "An investigation into the longevity of Cambridge sportsmen," *British Medical Journal*, vol. 1, no. 4865, pp. 773–777, 1954.

[41] C. D. Lee, X. Sui, S. P. Hooker, J. R. Hébert, and S. N. Blair, "Combined impact of lifestyle on cancer mortality in men," *Annals of Epidemiology*, vol. 21, no. 10, pp. 749–754, 2011.

[42] E. S. Ford, G. Zhao, J. Tsai, and C. Li, "Low-Risk lifestyle behaviors and all-cause mortality: findings from the national health and nutrition examination survey III mortality study," *American Journal of Public Health*, vol. 101, no. 10, pp. 1922–1929, 2011.

[43] H. M. Ahmed, M. J. Blaha, K. Nasir, J. J. Rivera, and R. S. Blumenthal, "Effects of physical activity on cardiovascular disease," *American Journal of Cardiology*, vol. 109, no. 2, pp. 288–295, 2012.

[44] Z. Radak, N. Hart, L. Sarga et al., "Exercise plays a preventive role against Alzheimer's disease," *Journal of Alzheimer's Disease*, vol. 20, no. 3, pp. 777–783, 2010.

Hindawi Publishing Corporation
Journal of Aging Research
Volume 2012, Article ID 321098, 10 pages
doi:10.1155/2012/321098

Research Article

Health-Related Social Control over Physical Activity: Interactions with Age and Sex

Kelly A. Cotter

Department of Psychology, Sacramento State University, 6000 J Street, Sacramento, CA 95819, USA

Correspondence should be addressed to Kelly A. Cotter, cotterk@csus.edu

Received 3 February 2012; Accepted 13 April 2012

Academic Editor: Iris Reuter

Despite the disease prevention benefits of engaging in life-long regular physical activity, many adults remain sedentary. The social environment provides an important context for health and health behavior across the lifespan, as well as a potential point of intervention for increasing physical activity. Self-reports of perceived social support, social strain, positive social control, and negative social control were examined for their cross-sectional relationships to physical activity frequency in purposive samples of younger and older adults ($N = 371$, ages from 18 to 97, 68% women). Hierarchical regression analyses revealed that perceived support and perceived strain were not correlated with physical activity. However, age and sex interacted with social control, such that more positive social control was associated with more frequent physical activity for younger men. Furthermore, more positive and negative social control were significantly associated with less frequent physical activity for older men, while social control was not associated with physical activity among women. While younger men may be encouraged toward healthier behaviors by positive social control messages, social control attempts may backfire when targeting older men. Implications for physical activity promotion are discussed.

1. Introduction

Engaging in regular physical activity is a powerful tool for maintaining health and reducing disability across the lifespan [1]. The Centers for Disease Control and Prevention (CDC, [2]) recommend that all adults, including older adults, should accumulate *at least* 150 minutes of moderate-intensity aerobic activity (e.g., brisk walking) or 75 minutes of vigorous-intensity aerobic activity (e.g., running) on most days of the week. An equivalent combination of moderate and vigorous aerobic activity is also acceptable. In addition to strengthening the cardiovascular system through aerobic activity, the CDC also recommends that adults participate in strength-building activity on at least two days per week. Importantly, these activity bouts can be broken into 10-minute sessions and spread throughout the week. Furthermore, these activity bouts do not have to be planned, structured exercise sessions; Leisure activities like gardening or bowling also "count" toward the accumulation of health-enhancing activity minutes, as long as the individual participating in the activity perceives it to be moderately or vigorously intense.

The CDC's physical activity recommendations are based on research demonstrating that physical activity effectively reduces risk for age-related diseases including diabetes, heart disease, cerebrovascular disease, osteoarthritis, and some cancers [3]. In addition, greater physical activity is associated with more strength and balance, relief of many disease symptoms, and increased longevity [3]. Due to its profound health effects, it is imperative to understand how to promote physical activity behavior across the lifespan. The social landscape provides one important context for physical activity behavior [1], and it also changes in meaningful ways for men and women as they age [4]. Thus, in the present study, I examined the direct and moderated relationships of physical activity to perceived social support, perceived social strain, positive social control, and negative social control.

Perceived social support, defined by Walen and Lachman as "one's perceived notion of the caring and understanding exhibited by the [social] network" [5, page 7], has a consistent positive influence on health and health behaviors [6].

On the other hand, perceived strain, defined as "individuals' general perception of the critical, irritating, and unreliable nature of their network" [5, page 7], typically has negative implications for health and health behaviors [7].

The structure of the social network, and thus the availability of support and strain, changes in meaningful ways as people age. The social network narrows in later life, such that older adults maintain or increase contact with intimates (including spouses and close friends) but reduce contact with acquaintances [8]. This pattern is consistent with socioemotional selectivity theory [4], which states that people are motivated by emotional goals when they perceive time as limited. Applying the sense of time-sensitivity to the lifespan, older adults (relative to younger adults) are more motivated to maintain bonds with sources of support and sever ties with sources of strain because older adults are closer to the end of the lifespan and thus more motivated to induce positive emotional states [8]. Therefore, older adults should report more support and less strain from their social partners than do younger adults. Consistent with this assertion, Walen and Lachman [5] found that older adults (age 60–75) reported more family support and less family strain than younger adults (age 25–39) in a sample of married or cohabitating adults. Furthermore, older men reported more support from their spouse than younger men [5].

In addition to predicting health directly, perceived support is also linked to greater physical activity participation [1]. Furthermore, although perceived strain is associated with poor health outcomes, it has a positive relationship with physical activity. For example, in a large national survey of adults aged 35 to 84 years, participants who reported more perceived strain also reported more frequent physical activity [1]. The relationship between perceived strain and physical activity was moderated by age, such that it was stronger for older adults. To explain the counterintuitive relationship between strain and physical activity, some authors have suggested that people may use social sanctions to elicit desired behavior in social partners [9]. These interactions, while potentially well intended and effective, may be perceived as straining and controlling. Indeed, health-related social control, broadly defined as the efforts of network members to change a target's health behavior [10], provides a potential pathway to health.

At the most basic level, health-related social control is theorized to act as the mechanism by which network members influence health, such that someone in the social network exerts social control, which causes the recipient to change his or her targeted health behavior, which in turn affects the recipient's health [11]. These control attempts can be perceived as positive (e.g., encouragement) or negative (e.g., criticism) [12]. For example, a person who wants to support a friend by including her in activities might invite her to a water aerobics class. This friend may interpret such support as encouragement for healthy behaviors (positive social control), or she might feel nagged to exercise (negative social control). Thus, social control may explain the relationship of perceived support to health behaviors, such that social control provides one of many strategies to offer support [1]. In fact, social control and general perceived support are moderately correlated [13].

It is important to point out that general perceived support is not the same concept as domain-specific social support [13]: general support refers to a person's global perceptions about his or her available supports, while domain-specific support refers to a person's perceptions of available support or actual support received given specific circumstances. Domain-specific support has received a lot of attention in the physical activity literature. Studies show that greater exercise-support or physical activity-support predict greater exercise or physical activity participation [14]. Domain-specific support is the same concept as positive social control (i.e., encouragement for physical activity participation), but the terms used to describe the concept have differed in the literature. I use the social control terminology in this paper.

An overwhelming majority of research has found that positive social control is linked to health behavior change [15], including physical activity [16], while negative social control is associated with resistance to behavior change [17]. However, the influence of social control may change across the life course. For example, Tucker et al. found that older adults reported fewer people in their networks who provided social control than younger adults [18]. Additionally, older adults who reported more positive affect from social control attempts were more likely to hide unhealthy behaviors from the social network [19]. Tucker et al. argue that older adults want to maintain harmony with the social network but may be unwilling to give up negative health behaviors, so instead they hide these behaviors [19]. This explanation is consistent with socioemotional selectivity theory, which states that older adults are more motivated than younger adults to satisfy emotional goals, like maintaining harmony with the social network [4]. Tucker and colleagues' results suggest that positive social control may not necessarily promote health behavior among older adults, particularly if they enjoy high-quality relationships [19].

In addition to being less effective for older adults than for younger adults, social control attempts may also be less effective for women than for men. Men are more likely than women to be the targets of social control in marriages [20], and wives are more successful social control agents than husbands [21]. Even outside of the marital relationship, social control attempts from the partner, family, and friends were effective at reducing men's smoking behavior over four months but had no effect on women's smoking [22]. Taken together, previous research suggests that social control may not be an effective strategy for changing the health behaviors of older adults or women. However, social control may be successful when targeting the health behaviors of younger men. Because younger men might react differently to social control than older men or women react, the potential interaction between age, sex, and social control was explored in the present study.

The present study was designed to examine the links between physical activity behavior, perceived support and strain, and health-related social control. As argued above, positive social control shares conceptual similarities to social support and may even act as a method of providing emotional, informational, and instrumental support [13]. While research has demonstrated the independence of social control from social support [13], social control has not yet been examined for its independence from social strain, despite conceptual similarities. In fact, Cotter and Lachman have argued that social partners may intend to be supportive with their health-related messages, but that recipients of those messages may perceive such interactions as straining [1]. Therefore, from the perspective of the target, strain may share an even stronger relationship to social control than support does. Examining all four forms of social influence together is imperative for informing health behavior interventions because this will identify which type has the strongest relationship to health behavior, and thus the most appropriate target for intervention.

Based on the results of previous studies, I predicted that more frequent physical activity would be associated with more perceived support and strain, and that the positive relationship of strain to physical activity would be stronger among older adults than among younger adults [1]. I also explored a potential interaction between age and sex in the context of health-related social control. Based on previous research revealing that social control was ineffective when targeting older adults [19] and women [22], I predicted that age and sex would moderate the relationship of social control to physical activity in the present study, such that more positive and less negative social control would be associated with more frequent physical activity for younger men, but that social control would not be related to physical activity for older adults and for women.

2. Method

2.1. Procedure. Data for the present study were simultaneously collected from two different sources: undergraduate psychology majors at a northern California university and community-dwelling older adults (who were recruited through flyers and presentations at local senior centers). Upon volunteering, participants were guided through an informed consent procedure. Next, participants completed the survey. A research assistant was available to answer questions as the participants completed the survey. Finally, participants were debriefed and given information about psychological and fitness resources. Undergraduates received course research credit for their participation, and older adults were entered into a raffle for one of fifteen $25 gift cards. The protocol received institutional approval for the ethical treatment of participants.

2.2. Participants. Participants were 371 noninstitutionalized, English-speaking adults ages from 18 to 97 ($M = 45.28$, SD = 26.93), who volunteered to participate in a paper-and-pencil survey. The sample recruited through the university ranged between 18 and 52 years old ($M = 22.02$, SD = 4.46), while the sample recruited through senior centers ranged between 46 and 97 years old ($M = 73.71$, SD = 10.72). Information regarding the demographic characteristics of race/ethnicity, marital status, education, and income was also collected (see Table 1), and each of these variables was dichotomized for analyses.

For the race/ethnicity variable, White participants comprised one group (51.3%) and all other races were combined to create the non-White group (48.7%). Marital status was recoded with one category of participants who were currently married (17%) and the second category with participants who were separated, divorced, widowed, or never married (83%). Education was dichotomized with one group of participants who had earned up to a high school diploma or GED (22.9%) and the other group who attended some college or higher education (77.1%). Finally, annual household income was recoded such that participants earning $20,000 or less comprised one group (50%) and participants earning $20,001 or more comprised the other group (50%).

The undergraduate sample reflects an accurate representation of the age and ethnicity of the undergraduate population of the university. However, women are overrepresented [23]. Older women, African Americans, and people of multiethnic heritage are overrepresented in the older adult sample compared to the population of American older adults [24, 25]. Furthermore, it should be presumed that the older adults sampled for the present study represent a relatively healthy and active segment of the older adult population. While participants were not asked specific questions regarding health conditions or pain; all were healthy enough to either attend classes at a university or to attend functions and activities at a community center.

2.3. Measures

2.3.1. Perceived Support and Strain. Responses to questions reflecting perceived social support (e.g., "How much do your friends really care about you?") and perceived social strain (e.g., "How often does your family criticize you?") from the spouse/partner, family, and friends were averaged across source [5], with higher scores reflecting more support ($M = 3.45$, SD = .48, $\alpha = .84$) or strain ($M = 1.92$, SD = .58, $\alpha = .85$). Although only 17% of the sample reported being married, most participants (54%) had a significant other to which they referred on items regarding partner support and strain. The range of scores in the present sample reflects the possible range (1 = Not at all to 4 = A lot) and published reliability (α range from .79 to .91) [5].

2.3.2. Social Control over Physical Activity. Social control over physical activity was measured as positive and negative social control from the spouse/partner, family, and friends. The positive social control scale (e.g., "How much does your partner encourage you to exercise?") was adapted from Sallis et al. [26], who reported high test-retest and internal consistency reliability for the measure. Responses to items were averaged, with higher scores reflecting more positive social control ($M = 2.39$, SD = .99, $\alpha = .97$). The range of scores in the present sample reflects the possible range (1 = Never to 5 = Very Often).

Table 1: Summary of participant characteristics.

Variable	Total Sample (N = 371)		Younger Adults (N = 205)		Older Adults (N = 166)	
	N	%	N	%	N	%
Sex						
Male	171	31.7	56	27.9	60	36.1
Female	252	68.3	145	72.1	106	63.9
Race/Ethnicity						
White	183	51.3	81	41.5	101	63.1
African American	41	11.5	17	8.7	24	15.0
Mexican American	43	12.0	31	15.9	11	6.9
Native American	7	2.0	4	2.1	3	1.9
Asian or Pacific Islander	46	12.9	42	21.2	4	2.5
Other	14	3.9	10	5.1	4	2.5
Multiracial	23	6.4	10	5.1	13	8.1
Marital Status						
Married	61	17.0	12	6.0	49	31.4
Separated	6	1.7	0	0	6	3.8
Divorced	42	11.7	0	0	42	26.9
Widowed	45	12.5	1	.5	43	27.6
Never Married	205	57.1	188	93.5	16	10.3
Education						
No school/some grade school	2	.6	0	0	2	1.3
Junior high school	7	2.0	0	0	7	4.6
Some high school	11	3.1	0	0	11	7.2
GED	4	1.1	0	0	4	2.6
High school diploma	26	7.3	0	0	26	17.1
1-2 years of college	130	36.7	109	54.2	20	13.2
3 or more years of college	64	18.1	54	26.9	10	6.6
Associate's degree	49	13.8	32	15.9	17	11.2
Bachelor's degree	27	7.6	6	3.0	21	13.8
Some graduate school	11	3.1	0	0	11	7.2
Master's degree	19	5.4	0	0	19	12.5
Professional degree	4	1.1	0	0	4	2.6
Annual Income						
Less than \$10,000	91	27.2	57	29.1	34	24.8
\$10,001–\$20,000	77	23.0	35	17.9	40	29.2
\$20,001–\$50,000	81	24.2	42	21.4	39	28.5
\$50,001–\$75,000	33	9.9	23	11.7	10	7.3
\$75,000 or more	53	15.8	39	19.9	14	10.2

Note: Age ranged from 18 to 42 (M = 21.76, SD = 3.56) for younger adults and from 46 to 97 (M = 73.44, SD = 11.02) for older adults.

The negative social control measure was created for the present study to measure negative social control from the spouse/partner, family, and friends aimed at promoting exercise behavior. Modeled after the positive social control measure [26], respondents were asked to report how often their spouse/partner, family members not including the spouse or partner, or friends did each of the following in the past month: nag you about exercise, demand that you exercise on recreational outings, demand that you discuss exercise, tell you ideas on how you can get more exercise, make negative comments about your physical appearance, pressure you to exercise, and make remarks about how much you should be exercising. Participants responded on a five-point scale (1 = Never, 2 = Rarely, 3 = Sometimes, 4 = Often, 5 = Very Often).

A principal components factor analysis was conducted on each subscale, and each subscale had an eigenvalue above 2. The factor solution for the partner domain included one factor that accounted for 53.74% of the variance with an eigenvalue of 3.76 (α = .84). The factor solution for the family domain included one factor that accounted for 55.56% of the variance with an eigenvalue of 3.89 (α = .86). The factor solution for the friend domain included one factor that accounted for 45.97% of the variance with an eigenvalue of 3.22 (α = .77). Responses were averaged across social partner for the present study, with possible scores ranging from 1 to 5. Higher scores reflect more negative social control (range = 1–5, M = 1.53, SD = .56, α = .89).

2.3.3. Physical Activity. Physical activity was measured with 9 items assessing participants' frequency of vigorous and moderate physical activity on a six-point scale ranging from 0 (Never) to 5 (Several times a week). Participants' scores reflected the possible range in the present sample. Based on Cotter and Lachman's measure [1], the setting in which the participant was most active (work, home, or leisure) comprised the participants' vigorous and moderate scores. In other words, the highest "moderate" score of the three settings was used as the indicator of frequency of moderate physical activity, and the highest "vigorous" score of the three settings was used as the indicator of frequency of vigorous physical activity. In this manner, if the participant performed regular activity in the home but not at work or for leisure, the respondent was still classified as regularly active. Next, the higher of the moderate versus vigorous scores was used as the indicator of total physical activity frequency (M = 3.89, SD = 1.59). Moderate (M = 3.77, SD = 1.63) and vigorous (M = 3.10, SD = 1.88) activity were highly correlated with each other in the present study: r = .66, P < .001. Cotter and Lachman [1] reported that calculating the physical activity score in this manner yields the best approximation possible to data from the Centers for Disease Control and Prevention [2], which recommends that adults accumulate at least 30 minutes of moderate aerobic activity on most days of the week or 20 minutes of vigorous aerobic activity three days per week.

3. Results

First, analyses were conducted on all variables to ensure normality of the distribution and reliability of measures. The negative social control measure was positively skewed and was transformed using a log 10 transformation. Next, exploratory analyses were conducted to determine whether there were different patterns of results based on different sources of influence (partner, family, and friends). Patterns were consistent between the partner, family, and friend domains. Thus, social influence variable scores were averaged across the domains, as described above in the measures section. All continuous independent variables were centered, and 2- and 3-way interaction terms were calculated by multiplying the centered support, strain, positive social control, or negative social control score by sex (0 = male, 1 = female) and age (0 = younger, 1 = older). Age was dichotomized at the mean of 45 years because the sampling technique led to a very bimodal distribution (n = 205 for younger adults, n = 166 for older adults, see Table 1).

Zero-order correlations between all variables were calculated and examined (see Table 2). Next, the direct and moderated cross-sectional relationships with physical activity frequency were examined in Hierarchical Multiple Regression (HMR) analyses using pairwise deletion and the MODPROBE procedure for SPSS developed by Hayes and Matthes [27]. Interactions with positive social control and negative social control were each examined in separate analyses in order to reduce problems associated with multicollinearity. Thus, age, sex, perceived support, perceived strain, positive social control, and negative social control were entered on Step 1 of both HMR analyses. Two-way age, sex, and *positive* social control interaction terms were entered on Step 2 of Model 1 (presented in the top half of Table 3), and two-way age, sex, and *negative* social control interaction terms were entered on Step 2 of Model 2 (presented in the bottom half of Table 3). The three-way interaction term age-by-sex-positive social control was entered on Step 3 of Model 1, and the three-way interaction term age-by-sex-by-negative social control was entered on Step 3 of Model 2. The Johnson-Neyman technique was used in follow-up analyses to examine regions of significance for the interactions [27].

Regression analyses revealed that none of the demographic characteristics had a significant relationship to physical activity when age and sex were in the models, so demographic characteristics were trimmed from the analyses to conserve statistical power. Furthermore, consistent with the bivariate relationships presented in Table 2, regression analyses revealed no statistically significant direct or moderated relationships between perceived social support and perceived social strain with physical activity. Thus, the three-way interaction terms of age-by-sex-by-perceived support and age-by-sex-by-perceived strain were also trimmed from the reported models to conserve power, but are available upon request. The results of the regression analyses are shown in Table 3, with interactions involving positive social control presented in the top half of the table and interactions involving negative social control presented in the bottom half of the table.

3.1. Positive Social Control. Variables in the model examining positive social control explained 20.1% of the total variance in physical activity frequency, F (10, 262) = 6.60, P < .001, and revealed a significant 3-way interaction between age, sex, and positive social control (β = .27, P = .05, see top half of Table 3) even after controlling for the relationships of perceived support, perceived strain, and negative social control. To plot the interaction, the data were split by sex and the 2-way interactions of age and positive social control were examined separately for men and women. The figures show predicted regression lines for younger and older adults at one standard deviation below (low) and one standard deviation above (high) the mean of positive social control. As shown in Figure 1, the 2-way age-by-positive social control interaction was significant among men, ΔR^2 = .10, F (1, 73) = 9.85, P < .001. Follow-up analyses used the Johnson-Neyman technique to determine regions of significance within the interactive effect [27]. Specifically, the effect of positive social control at each level of age (younger versus older) was examined among men and women. The follow-up analyses revealed that more positive social control was associated with more frequent physical activity for younger men (t = 1.67,

Table 2: Correlations between all variables.

		1	2	3	4	5	6	7	8	9	10	11
1	Age	—	−.09	**−.22**	**.34**	**−.21**	−.07	−.05	**−.18**	−.09	−.10	**−.38**
2	Sex		—	−.00	*−.10*	.01	−.07	**.17**	*−.13*	.07	−.08	−.03
3	Race			—	−.08	−.05	−.06	−.08	**.25**	−.03	.08	.03
4	Marital status				—	.00	**.19**	.07	.09	*.12*	−.01	**−.16**
5	Education					—	*.13*	.05	−.04	*.11*	.00	**.18**
6	Income						—	*.12*	−.05	−.01	**−.17**	.01
7	Support							—	−.08	**.34**	.04	.11
8	Strain								—	**.17**	**.52**	.03
9	PSC									—	**.28**	.12
10	NSC										—	−.01
11	Physical activity											—

Note: $P \le .05$, $P < .01$. Age was dichotomized such that 0 = younger than 45 and 1 = older than 45, sex was dichotomized such that 0 = male and 1 = female, race/ethnicity was dichotomized such that 0 = White and 1 = all other races, marital status was dichotomized such that 0 = separated, divorced, widowed, or never married, and 1 = currently married, education was dichotomized such that 0 = up to a high school diploma or GED and 1 = attended some college or higher education, and annual household income was dichotomized such that 0 = \$20,000 or less and 1 = \$20,001 or more. PSC refers to positive social control and NSC refers to negative social control.

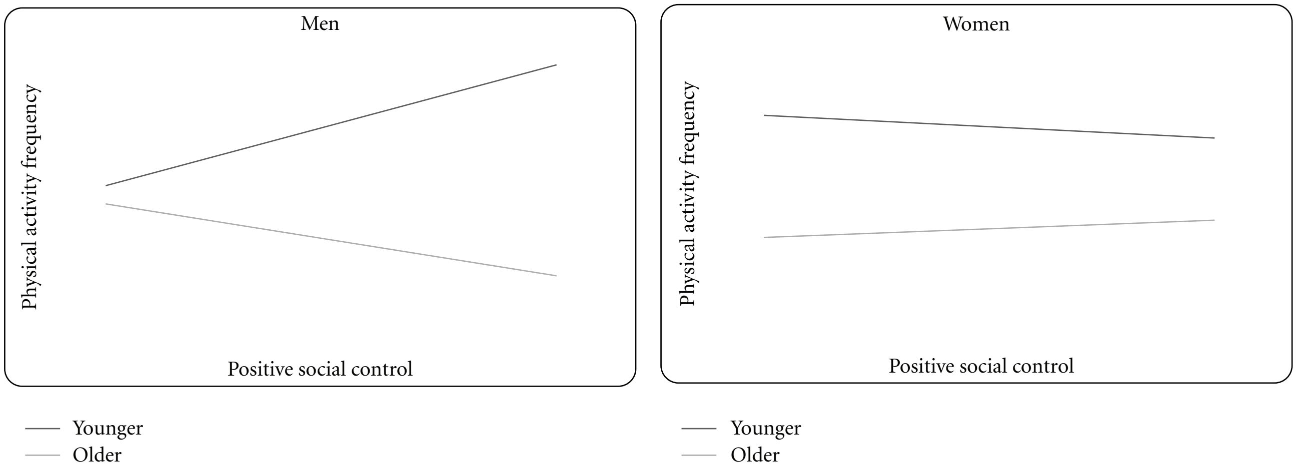
Table 3: Summary of two HMR analyses predicting physical activity frequency.

	Step 1			Step 2			Step 3		
	B	SE	β	B	SE	β	B	SE	β
Age	−1.25	.18	−.39**	−1.20	.32	−.38**	−1.24	.32	−.39**
Sex	−.30	.20	−.09	−.29	.27	−.09	−.30	.27	−.09
Support	.26	.20	.08	.34	.20	.10*	.34	.20	.11
Strain	−.07	.18	−.02	−.05	.19	−.02	−.00	.19	-.00
PSC	.13	.10	.08	.37	.21	.23	.74	.28	.46**
NSC	−.80	.80	−.07	−.59	.80	−.05	−.55	.80	−.05
PSC *Age				−.46	.19	−.20*	−1.03	.35	−.45**
PSC *Sex				−.04	.21	−.02	−.52	.32	−.26
Age *Sex				−.07	.39	−.02	−.01	.39	−.00
PSC *Age *Sex							.82	.42	.27*
Change stats	$\Delta R^2 = .17$, $F\,(6, 266) = 9.16^{**}$			$\Delta R^2 = .02$, $F(3, 263) = 1.99^{+}$			$\Delta R^2 = .01$, $F\,(1, 262) = 3.87^{*}$		
Age	−1.25	18	−.39**	−1.15	.32	−.36**	−1.05	.32	−.33**
Sex	−.30	.20	−.09	−.25	.27	−.07	−.15	.27	−.05
Support	.26	.20	.08	.30	.20	.09	.33	.20	.10
Strain	−.07	.18	−.02	−.00	.19	−.00	.10	.19	.04
PSC	.13	.10	.08	.15	.10	.09	.15	.10	.09
NSC	−.80	.80	−.07	−.21	1.57	−.02	4.04	1.99	.34*
NSC *Age				−2.01	1.45	−.13	−8.14	2.32	−.52**
NSC *Sex				.72	1.49	.04	−5.33	2.33	−.33*
Age*Sex				−.12	.40	−.04	−.13	.39	−.04
NSC*Age*Sex							9.91	2.97	.41**
Change stats	$\Delta R^2 = .17$, $F\,(6, 265) = 9.12^{**}$			$\Delta R^2 = .01$, $F(3, 262) = .93^{+}$			$\Delta R^2 = .03$, $F\,(1, 261) = 11.16^{**}$		

Note: $^{+}P < .10$, $^{*}P < .05$, $^{**}P < .01$. PSC refers to positive social control, NSC refers to negative social control, *age refers to the interaction with age, and *sex refers to the interaction with sex.

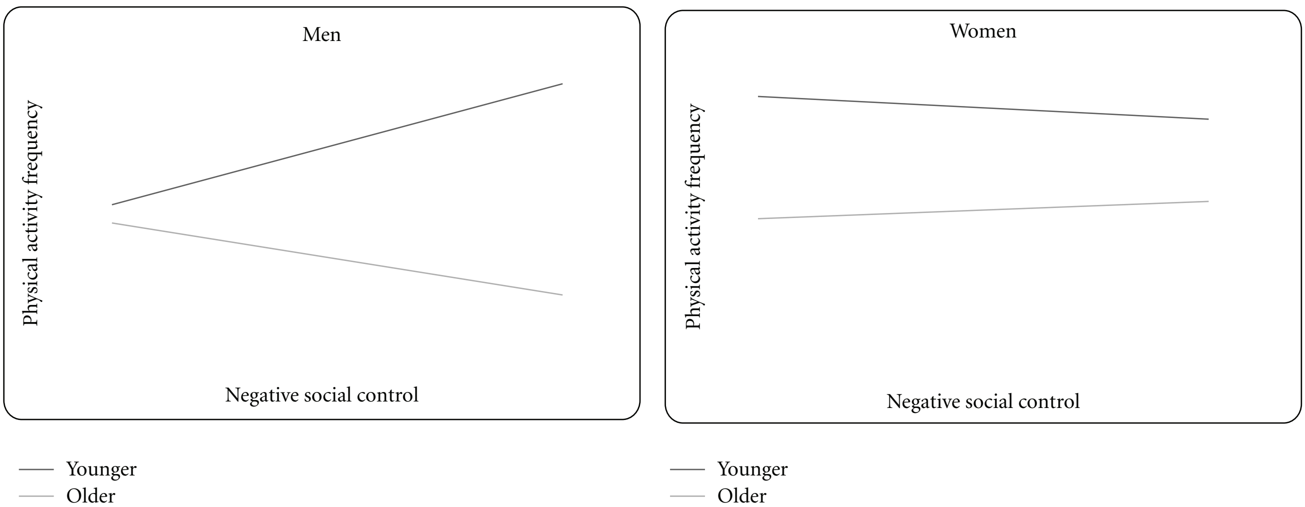
$P = .10$), while more positive social control was associated with significantly less frequent physical activity for older men ($t = -2.30$, $P = .03$). The interaction was not significant for women, $\Delta R^2 = .001$, $F\ (1, 174) = .31$, $P = .58$. Instead, only the direct relationship of age and physical activity was statistically significant, such that younger age was related to more frequent physical activity ($\beta = -.37$, $P < .001$).

3.2. Negative Social Control. Variables in the model examining negative social control explained 18.3% of the total



(a) (b)

Figure 1: (a) The interaction of age and positive social control on physical activity for men. (b) The interaction of age and positive social control on physical activity for women. *Note:* the figures show predicted regression lines for one standard deviation above (high) and one standard deviation below (low) the mean of positive social control. The simple slope is statistically significant for older men.



(a) (b)

Figure 2: (a) The interaction of age and negative social control on physical activity for men. (b) The interaction of age and negative social control on physical activity for women. *Note:* the figures show predicted regression lines for one standard deviation above (high) and one standard deviation below (low) the mean of negative social control. The simple slope is statistically significant for older men.

variance in physical activity frequency, $F\,(10, 261) = 7.09$, $P < .001$, and revealed a significant 3-way interaction between age, sex, and negative social control ($\beta = .41$, $P = .001$, see bottom half of Table 3) even after controlling for the relationships of perceived support, perceived strain, and positive social control. The interaction was plotted following the same method described above (see Figure 2), and further analysis using the Johnson Neyman technique [27] revealed that the 2-way age-by-negative social control interaction was significant among men, $\Delta R^2 = .09$, $F\,(1, 73) = 8.57$, $P = .005$, such that more negative social control was associated with less frequent physical activity for older men ($t = -2.52$, $P = .01$), while negative social control was not significantly associated with physical activity for younger men ($t = .90$, $P = .37$). Again here, the interaction was not significant for women, $\Delta R^2 = .003$, $F(1, 174) = .66$, $P = .42$. Instead, only the direct relationship of younger age and more frequent physical activity was statistically significant for women ($\beta = -.38$, $P < .001$).

4. Discussion

Younger age was associated with more frequent physical activity in all analyses. This is consistent with a large body of research demonstrating younger adults as more active than older adults [28]. Younger adults are less likely than older adults to suffer from disabling conditions [29] and are more aware of the benefits of exercise [30]. Thus, younger adults may have more opportunity to be active.

While the main effect of age on physical activity is important for public health intervention, the present study is among the first to examine the interactive effect of age, sex, and social control on physical activity. As predicted, age and sex moderated the relationship of social control to physical activity, such that more positive social control was associated with more frequent physical activity for younger men (albeit at trend-level statistical significance). These results are consistent with results from a sample of men living with HIV, demonstrating that positive social control from friends, family members, and romantic partners was associated with behavior change [17]. Because the current data are cross-sectional, we cannot determine if young men engage in health behaviors in response to social control attempts or, alternatively, if they receive social control in response to health behaviors. However, based on previous experimental research where social control predicted health behavior change [31], the social control to behavior direction seems more likely than the opposite.

In contrast to younger men, older men in the present study reported significantly less physical activity when they perceived more social control. These results do not support my hypothesis that social control would not affect older men. This hypothesis was based on a study from Tucker and colleagues, who found that older adults who reported more positive affect from social control attempts were more likely to hide unhealthy behaviors from the social network [19]. Consistent with socioemotional selectivity theory [4], Tucker et al. argued that older adults want to maintain harmony with the social network, but may be unwilling to give up negative health behaviors, so instead they hide these behaviors from their loved ones.

Alternatively, older men may suffer from more health conditions that benefit from engaging in physical activity. For example, older men are more likely than younger men to suffer from heart disease [32], a chronic illness that can be controlled through regular physical activity [33]. Social partners may engage in health-related social control in an attempt to encourage illness-management [13], but heart disease also makes physical activity more difficult to complete [33]. Thus older men may actively refrain from engaging in this health behavior, despite the social control attempts of their network members, because they are unable or unwilling to begin or maintain a physical activity regimen. In addition to health, response bias provides another potential explanation for the negative relationship between physical activity and social control: older men may perceive more social control from their social partner, or they may be more willing to report social control than younger men [20].

Consistent with previous research [22] and with hypotheses, social control had no relationship to physical activity among women in the present study. Westmaas et al. suggest that receiving social control may undermine women's perceptions of themselves as the health-keepers of their families and as positive role models for health behaviors [22]. Westmaas et al. explain that this psychological burden may interfere with women's ability to change their health behavior. Thus, women may need to bolster their cognitive and emotional resources (i.e., self-efficacy) before attempting a physical activity behavior change. Because older adults have low confidence in their ability to adopt and maintain a physically active lifestyle [34], they may also need to strengthen their self-efficacy before becoming active. Once they are confident in their physical abilities, positive social control may further enhance self-efficacy and promote activity [35].

While the relationship between social control and self-efficacy still needs to be examined empirically, there is evidence for a relationship between exercise self-efficacy and *exercise-specific* social support (which I have argued is the same concept as positive social control for exercise) [14], as well as exercise self-efficacy and *general* social support [36] among older adults. Furthermore, positive and negative aspects of social relations may have different relationships to outcomes depending on whether they are examined cross-sectionally or longitudinally [37]. Therefore, future research should examine physical activity's longitudinal relationship to both positive and negative social control, as well as social control's cross-sectional and longitudinal relationship to exercise self-efficacy.

Contrary to predictions, general perceived support and strain were not associated with physical activity in the current investigation. These results are inconsistent with a study demonstrating that more perceived support and strain were associated with more frequent physical activity among adults [1]. The discrepancy here may be attributed to study design and statistical power. Cotter and Lachman examined data from over 3,000 participants, whereas I examined data from approximately 300 participants in the current investigation. It is possible that with more statistical power the relationships found in the current investigation would have reached statistical significance. In fact, effect sizes for the relationships in the present study, while not statistically significant, are similar to the effect sizes reported by Cotter and Lachman.

Perceived support was moderately related to positive social control in bivariate analyses in the present study, consistent with results reported by Helgeson et al. [13] and McAuley et al. [14]. Additionally, perceived strain was moderately related to both negative social control and positive social control. These results are consistent with assertions that strain may share a stronger relationship to social control than support does, particularly from the perspective of the recipient of social influence [1]. Social partners, then, should be encouraged to be careful to remain positive in their interactions with social control targets. Positive social control is more likely to elicit positive affect, which leads to behavior change without also eliciting negative affect [16]. Thus, positive social control attempts have greater potential for success with fewer negative repercussions.

When evaluating suggestions based on the present results, it is important to consider the study's strengths and limitations. Regarding the limitations, variables were all examined using a self-report method. Thus, some of the shared variance can be accounted for by the method of data collection. Second, the present study may have lacked sufficient power to corroborate the statistical significance of relationships that have been found using larger samples [1]. Most importantly, the present investigation is based on cross-sectional data. Thus, conclusions regarding causal relationships cannot be made. Furthermore, age differences may be attributed to cohort effects rather than developmental effects. For example, the older adults sampled, particularly the older women, may have approached physical activity differently than the younger adults due to generational differences in attitudes, beliefs, and normative behaviors. While care was taken when collecting the present data to describe physical activity in detail at the outset, older and younger adults may have responded to questions regarding physical activity according to preconceived definitions and thus the results must be interpreted with caution.

The current study also had a number of strengths. First, perceived support and strain and positive and negative social control were all examined simultaneously, making comparisons between different types of social interaction possible. Indeed, this was the first study to examine perceived strain's relationship to social control. Results from the present investigation suggest that social control may be a more efficacious avenue to explore for physical activity promotion than general support and strain. Second, the use of younger and older adult samples allowed for age comparisons, which revealed different patterns of relationships between variables for younger and older cohorts. Finally, the present sample contained a high percentage of minority participants who are typically underrepresented in research. Thus, the results of the present study are generalizable to a broad population of healthy younger and older adults.

The results of the current investigation demonstrate how and for whom social control is associated with physical activity. However, longitudinal work must be completed in order to determine the causal direction of these relationships. This is especially important given that previous research shows that social influence variables have different cross-sectional and longitudinal relationships to outcomes [36]. Furthermore, experience time sampling designs, such as daily diary studies, would be useful to determine how daily interactions influence physical activity on that day or in a given week. Daily diary designs would be particularly informative for understanding how, when, and from whom social control might promote physical activity [38]. Finally, self-efficacy plays an important role in behavior adoption [39] and is influenced by the social environment [40]. Therefore, future work should examine the relationship of social control to self-efficacy.

In conclusion, health professionals and intimate social partners should be discouraged from using negative social control strategies because of the potential to induce negative affect, because these strategies may be ineffective for women and younger men, and because these strategies may be counterproductive for older men. Instead, it might be more appropriate to use positive social control strategies, particularly for younger male targets, in order to maintain feelings of support and positive affect while encouraging health behavior change. On the other hand, alternative strategies should be pursued for improving the health and fitness of women and older adults. For example, bolstering self-efficacy may provide an efficacious strategy within these populations.

Acknowledgments

Special thanks are due to the Active Aging Lab for their support with data collection. This paper was partially supported by the Sacramento State University 2009-2010. Research and Creative Activity Award Program Summer Fellowship Grant.

References

[1] K. A. Cotter and M. E. Lachman, "No strain, No Gain: psychosocial predictors of physical activity across the adult lifespan," *Journal of Physical Activity and Health*, vol. 7, no. 5, pp. 584–594, 2010.

[2] Centers for Disease Control and Prevention, "Physical activity for everyone," 2011, http://www.cdc.gov/nccdphp/dnpa/physical/everyone/recommendations/.

[3] American College of Sports Medicine, *ACSM's Guidelines for Exercise Testing and Prescription*, Lippincott, Williams, and Wilkins, Philadelphia, Pa, USA, 2000.

[4] L. L. Carstensen, D. M. Isaacowitz, and S. T. Charles, "Taking time seriously: a theory of socioemotional selectivity," *American Psychologist*, vol. 54, no. 3, pp. 165–181, 1999.

[5] H. R. Walen and M. E. Lachman, "Social support and strain from partner, family, and friends: costs and benefits for men and women in adulthood," *Journal of Social and Personal Relationships*, vol. 17, no. 1, pp. 5–30, 2000.

[6] S. Cohen, "Social relationships and health," *American Psychologist*, vol. 59, no. 8, pp. 676–684, 2004.

[7] T. C. Antonucci, H. Akiyama, and J. E. Lansford, "Negative effects of close social relations," *Family Relations*, vol. 47, no. 4, pp. 379–384, 1998.

[8] L. L. Carstensen, "Social and emotional patterns in adulthood: support for socioemotional selectivity theory," *Psychology and Aging*, vol. 7, no. 3, pp. 331–338, 1992.

[9] N. Krause, L. Goldenhar, J. Liang, I. G. Jay, and D. Maeda, "Stress and exercise among the Japanese elderly," *Social Science and Medicine*, vol. 36, no. 11, pp. 1429–1441, 1993.

[10] K. S. Rook, "Support, companionship, and control in older adults' social networks: implications for well-being," in *Handbook of Communication and Aging Research*, J. F. Nussbaum and J. Coupland, Eds., pp. 437–463, Lawrence Erlbaum Associates, Inc, Hillsdale, NJ, USA, 1995.

[11] D. Umberson, "Family status and health behaviors: social control as a dimension of social integration," *Journal of Health and Social Behavior*, vol. 28, no. 3, pp. 306–319, 1987.

[12] M. A. Lewis and R. M. Butterfield, "Social control in marital relationships: effect of one's partner on health behaviors," *Journal of Applied Social Psychology*, vol. 37, no. 2, pp. 298–319, 2007.

[13] V. S. Helgeson, S. A. Novak, S. J. Lepore, and D. T. Eton, "Spouse social control efforts: relations to health behavior and well-being among men with prostate cancer," *Journal of Social and Personal Relationships*, vol. 21, no. 1, pp. 53–68, 2004.

[14] E. McAuley, G. J. Jerome, S. Elavsky, D. X. Marquez, and S. N. Ramsey, "Predicting long-term maintenance of physical

activity in older adults," *Preventive Medicine*, vol. 37, no. 2, pp. 110–118, 2003.

[15] M. Logic, M. A. Okun, and J. A. Pugliese, "Expanding the mediational model of the effects of health-related social control," *Journal of Applied Social Psychology*, vol. 39, no. 6, pp. 1373–1396, 2009.

[16] M. M. Franks, M. A. P. Stephens, K. S. Rook, B. A. Franklin, S. J. Keteyian, and N. T. Artinian, "Spouses' provision of health-related support and control to patients participating in cardiac rehabilitation," *Journal of Family Psychology*, vol. 20, no. 2, pp. 311–318, 2006.

[17] E. Fekete, T. R. Geaghan, and J. A. Druley, "Affective and behavioural reactions to positive and negative health-related social control in HIV+ men," *Psychology and Health*, vol. 24, no. 5, pp. 501–515, 2009.

[18] J. S. Tucker, D. J. Klein, and M. N. Elliott, "Social control of health behaviors: a comparison of young, middle-aged, and older adults," *Journals of Gerontology—Series B*, vol. 59, no. 4, pp. P147–P150, 2004.

[19] J. S. Tucker, M. Orlando, M. N. Elliott, and D. J. Klein, "Affective and behavioral responses to health-related social control," *Health Psychology*, vol. 25, no. 6, pp. 715–722, 2006.

[20] D. Umberson, "Gender, marital status and the social control of health behavior," *Social Science and Medicine*, vol. 34, no. 8, pp. 907–917, 1992.

[21] M. A. Lewis, R. M. Butterfield, L. A. Darbes, and C. Johnston-Brooks, "The conceptualization and assessment of health-related social control," *Journal of Social and Personal Relationships*, vol. 21, no. 5, pp. 669–687, 2004.

[22] J. L. Westmaas, T. C. Wild, and R. Ferrence, "Effects of gender in social control of smoking cessation," *Health Psychology*, vol. 21, no. 4, pp. 368–376, 2002.

[23] Voluntary System of Accountability Program College Portrait, "California State University, Sacramento college portrait," 2011, http://wwwcollegeportraits.org/CA/CSUS/characteristics.

[24] L. M. Howden and J. A. Meyer, "Age and sex composition: 2010," Census Briefs, 2010, http://www.census.gov/prod/cen2010/briefs/c2010br-03.pdf.

[25] J. J. Pirkl, "The demographics of aging...," 2011, http://www.transgenerational.org/aging/demographics.htm#Characteristics.

[26] J. F. Sallis, R. M. Grossman, R. B. Pinski, T. L. Patterson, and P. R. Nader, "The development of scales to measure social support for diet and exercise behaviors," *Preventive Medicine*, vol. 16, no. 6, pp. 825–836, 1987.

[27] A. F. Hayes and J. Matthes, "Computational procedures for probing interactions in OLS and logistic regression: SPSS and SAS implementations," *Behavior Research Methods*, vol. 41, no. 3, pp. 924–936, 2009.

[28] Centers for Disease Control and Prevention, "Public health and aging: trends in aging—United States and worldwide," *Morbidity & Mortality Weekly Report*, vol. 52, no. 6, pp. 101–106, 2003.

[29] J. Cohen-Mansfield, M. S. Marx, and J. M. Guralnik, "Motivators and barriers to exercise in an older community-dwelling population," *Journal of Aging and Physical Activity*, vol. 11, no. 2, pp. 242–253, 2003.

[30] A. C. King, S. N. Blair, D. E. Bild et al., "Determinants of physical activity and interventions in adults," *Medicine and Science in Sports and Exercise*, vol. 24, no. 6, pp. S221–S236, 1992.

[31] M. A. Lewis and R. M. Butterfield, "Antecedents and reactions to health-related social control," *Personality and Social Psychology Bulletin*, vol. 31, no. 3, pp. 416–427, 2005.

[32] Federal Interagency Forum on Aging-Related Statistics, *Older Americans 2010: Key Indicators of Well-Being*, Federal Interagency Forum on Aging-Related Statistics, Washington DC, USA, 2010.

[33] Centers for Disease Control and Prevention, "Know the facts about heart disease," 2012, http://www.cdc.gov/heartdisease/docs/ConsumerEd_HeartDisease.pdf.

[34] F. D. Lees, P. G. Clark, C. R. Nigg, and P. Newman, "Barriers to exercise behavior among older adults: a focus-group study," *Journal of Aging and Physical Activity*, vol. 13, no. 1, pp. 23–33, 2005.

[35] J. S. Tucker and S. L. Anders, "Social control of health behaviors in marriage," *Journal of Applied Social Psychology*, vol. 31, no. 3, pp. 467–485, 2001.

[36] K. A. Cotter and A. M. Sherman, "Love hurts: the influence of social relations on exercise self-efficacy for older adults with osteoarthritis," *Journal of Aging and Physical Activity*, vol. 16, no. 4, pp. 465–483, 2008.

[37] K. S. Rook, "The negative side of social interaction: impact on psychological well-being," *Journal of Personality and Social Psychology*, vol. 46, no. 5, pp. 1097–1108, 1984.

[38] M. R. Mehl and T. S. Conner, *Handbook of Research Methods for Studying Daily Life*, The Guilford Press, New York, NY, USA, 2012.

[39] A. Bandura, *Self-Efficacy: the Exercise of Control*, W. H. Freeman, New York, NY, USA, 1997.

[40] E. McAuley, G. J. Jerome, D. X. Marquez, S. Elavsky, and B. Blissmer, "Exercise self-efficacy in older adults: social, affective, and behavioral influences," *Annals of Behavioral Medicine*, vol. 25, no. 1, pp. 1–7, 2003.

Hindawi Publishing Corporation
Journal of Aging Research
Volume 2012, Article ID 172492, 9 pages
doi:10.1155/2012/172492

Review Article

Role of Exercise Therapy in Prevention of Decline in Aging Muscle Function: Glucocorticoid Myopathy and Unloading

Teet Seene and Priit Kaasik

Institute of Exercise Biology and Physiotherapy, University of Tartu, Ravila 14a, 50411 Tartu, Estonia

Correspondence should be addressed to Teet Seene, teet.seene@ut.ee

Received 1 December 2011; Accepted 14 May 2012

Academic Editor: Barbara Tettenborn

Changes in skeletal muscle quantity and quality lead to disability in the aging population. Physiological changes in aging skeletal muscle are associated with a decline in mass, strength, and inability to maintain balance. Glucocorticoids, which are in wide exploitation in various clinical scenarios, lead to the loss of the myofibrillar apparatus, changes in the extracellular matrix, and a decrease in muscle strength and motor activity, particularly in the elderly. Exercise therapy has shown to be a useful tool for the prevention of different diseases, including glucocorticoid myopathy and muscle unloading in the elderly. The purpose of the paper is to discuss the possibilities of using exercise therapy in the prevention of glucocorticoid caused myopathy and unloading in the elderly and to describe relationships between the muscle contractile apparatus and the extracellular matrix in different types of aging muscles.

1. Introduction

Aging is a multifactorial process influenced by biological, physiological, psychological, and social changes. The biological and physiological changes are primarily associated with a decline in muscle mass, strength, endurance and the inability to maintain balance [1–3]. Physical risk factors for falling, such as muscle weakness and an inability to maintain static or dynamic balance, lead to severe injury in the elderly [3]. Changes in skeletal muscle quantity and quality lead to disability in the aging population [4].

The rate of muscle loss has been estimated to range from 1% to 2% per year past the age of 50, as a result of which 25% of people under the age of 70 and 40% over the age of 80 are sarcopenic [5, 6]. In both young and aged skeletal muscle, it has been shown that oxidative stress increases in response to unloading [7] and may have an important role in mediating muscle atrophy [8]. It has also been proposed that changes caused by aging and unloading are muscle specific [9]. Muscle unloading results in a decrease in the number of myonuclei and an increase in the number of apoptotic myonuclei in skeletal muscle [10]. Heat-shock protein (HSP) 70 inhibits caspase-dependent and caspase-independent apoptotic pathways and may function in the regulation of muscle size by inhibition of necrotic muscle fiber distribution and apoptosis in aged muscle [11, 12]. The decline of muscle mass is primarily caused by type II fiber atrophy and loss in the number of muscle fibers. Increased variability in fiber size, accumulation of nongrouping, scattered and angulated fibers, and expansion of extracellular space are characteristic to muscle atrophy [13, 14]. Loss of fiber number as well as decreased production of anabolic hormones, for example, testosterone, growth hormone, insulin-like growth factor 1 (IGF-1), and an increase in the release of catabolic agents are principal causes of sarcopenia, and interleukin-6 has also been shown to amplify the rate of muscle wasting [15, 16].

Aging skeletal muscle becomes less powerful, fat is redistributed from the depot to muscle [17], and altered collagen synthesis and posttranslational changes in the structure of collagen reduce the elasticity of ligaments [18, 19].

The properties of muscle strength and stiffness that control balance between the ability of muscle fibers to resist stretching depend on the degree of cross-linking of collagen molecules. With age, the number of cross-links increases and makes the collagen fibers too stiff for optimal function [20].

Skeletal muscle reloading after unloading has been shown to increase the recovery of motor activity, which is as fast as the recovery of muscle strength, but mechanical properties depend on the metabolism and regeneration of the muscle structures from disuse atrophy [4]. The qualitative remodeling of contractile proteins plays a certain role in impaired locomotion and general weakness in aging. Thus, when atrophic muscle becomes active again, muscle mass increases in a relatively short period of time but the recovery of muscle strength takes much longer [21].

Dexamethasone treatment increased aging muscle wasting much more than in the young [22, 23] and the main reason is the loss of myofibrillar proteins from muscle [22, 24]. The catabolic action of glucocorticoids on skeletal muscle was found to depend on the functional activity of muscle [25, 26]. Exercise with simultaneous glucocorticoid treatment is an effective measure in retarding skeletal muscle atrophy [27, 28] and provides protection against one of the major effects of glucocorticoid-muscle wasting [29]. The search for possibilities to rehabilitate the loss of physical function by exercise therapy in the elderly to prevent diseases is one of the challenges nowadays caused by the increase in the number of aging people in the society. The capacity to evoke structural and functional rearrangements in aging skeletal muscle depends on the oxidative potential of the fibers [30]. The integral indicator of muscle protein metabolism, the turnover rate, provides a mechanism by which strength exercise can change the renewal of contractile proteins in accordance with the needs of muscle contractile apparatus [31]. As oxidative capacity of skeletal muscle decreases in the elderly, endurance exercises seems to be effective in its restoration as it stimulates mitochondrial biogenesis and improves their functional parameters [32, 33]. Both, strength and endurance exercise seem to be promising tools for aging-related disease prevention.

In the present paper, we will discuss recent evidence of exercise therapy in the prevention of glucocorticoid caused myopathy and in case of skeletal muscle unloading, what is characteristic in aging population. We will describe the relationships between the muscle contractile apparatus and the extracellular matrix (ECM) in different types of aging muscle and changes in muscle strength, endurance, and motor activity.

2. Glucocorticoid-Caused Myopathy in Aging Muscle

The anti-inflammatory effect of glucocorticoids is the reason for their wide use in various clinical scenarios. A side effect of glucocorticoids is muscle atrophy (Figure 1). It is well known that glucocorticoid-caused myopathy as well as Cushing's disease lead to a marked reduction in muscle mass, wasting of muscle, loss of strength, and selective atrophy of fast-twitch (FT) muscle fibers [34].

Aging-caused sarcopenia is associated with muscle weakness and impaired locomotion. Dexamethasone treatment significantly decreases muscle strength and motor activity of laboratory animals [23] and humans [22]. The reduced muscle mass in aging and dexamethasone treatment reflect a loss of myofibrillar proteins [4, 22, 24]. In both laboratory animals and humans, the synthesis rate of myofibrillar but not of sarcoplasmatic proteins decreases with age [22]. The treatment of adult and aged laboratory animals with dexamethasone leads to muscle wasting, but this effect was much more rapid in aged animals [35]. One of the reasons for this is that glucocorticoids decrease the stimulatory effect of insulin and IGF-1 in the skeletal muscle of old rats twice as much as in adults [36] and increase the expression of myostatin, a negative regulator of skeletal muscle [37]. In FT muscle fibers, an excess of glucocorticoids causes a breakdown of thick and thin myofilaments and disintegration of individual myofibrils [38].

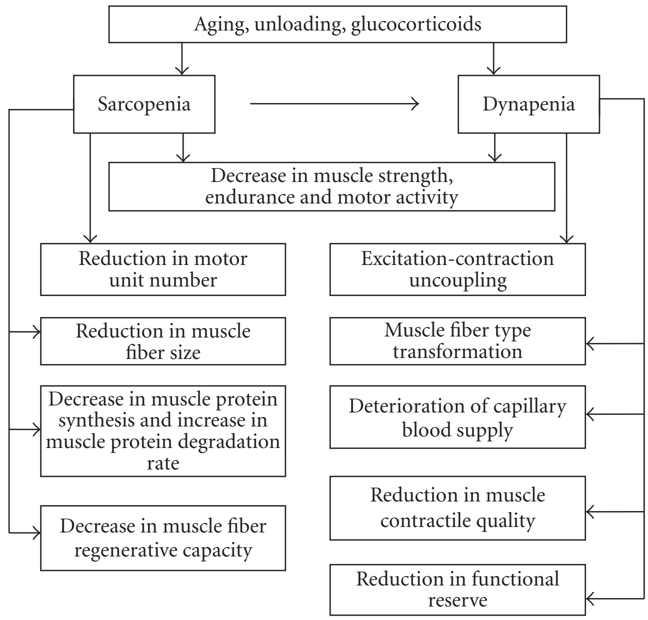


Figure 1: Effects of aging, unloading, and glucocorticoid treatment on skeletal muscle quantity and quality.

2.1. Role of Cellular and Extracellular Compartments in Changes of Muscle Strength and Motor Activity in Aging Myopathic Muscle. One of the important consequences of aging is impaired locomotion and general weakness [22]. Daily motor activity of old rats has a tendency to decrease in comparison with young rats. Dexamethasone treatment significantly reduced daily motor activity in both young- and old-age groups [39]. It has been shown there is a decline in muscle strength in atrophic muscle [40]. The qualitative remodeling of contractile proteins probably plays a certain role in this. Dexamethasone treatment decreased grip strength in both age groups but significantly more in the old than in the young group [23, 39]. It seems that in the everyday life of senescent rats, a decrease in muscle strength plays a more important role than daily motor activity.

The intensity of regeneration depends on the number of satellite cells under the basal lamina of muscle fibers, the size of the muscle, the type of injury, and the twitch characteristics of the muscle [30, 41–43]. Autografting of gastrocnemius muscle in old animals shows that regeneration proceeds significantly more slowly in comparison with young animals. Dexamethasone treatment decreased regeneration capacity both in young and old animals. Slower regeneration in old animals and after dexamethasone treatment in young and old groups is in good correlation with the decreased number of satellite cells [39]. Previous work has shown that dexamethasone treatment caused destructive changes in satellite cells on the ultrastructural level, which are similar to mother cell damage [38]. Myosin heavy chain (MyHC) composition during regeneration shifts from fast to slower type and this process is regulated by the cycle of denervation and reinnervation in regenerating skeletal muscle fibers [41].

Dexamethasone treatment led to quite similar results both in the young and the old but these changes are more significant in the aging group. Both aging and dexamethasone induced sarcopenic muscles have diminished regenerative capacity [39].

An excess of glucocorticoids has some similarities in its effect on the intracellular and extracellular compartments of skeletal muscle, for example, the decreased synthesis of proteins [34, 44]. The downregulation of collagen synthesis during dexamethasone administration shows that ECM components decrease [44]. As shown previously [38], MyHC synthesis rate only decreased in FT muscles, while the expression of collagen I, III, and IV mRNA decreased in both FT and slow-twitch (ST) muscles. It seems that dexamethasone treatment similarly influences fibril- and network-forming collagen expression in ST and FT muscles but differs at this point from contractile protein myosin synthesis, which was depressed only in FT muscles [45].

The second principal difference between contractile proteins and ECM during an excess of glucocorticoids is the degradation rate of proteins. Dexamethasone-increased the degradation of contractile proteins in skeletal muscle about two times but the expression of matrix metalloproteinase-2 (MMP-2) mRNA did not simultaneously significantly change although the degradation of collagens occurs mainly through MMP activity [46]. This is surprising as it was shown earlier that ST muscles contain significantly more collagen than FT muscles [47].

The concentration of endomysial collagen is higher around FT fibers [48], but as the downregulation of synthesis and unchanged degradation of type IV collagen are very similar to fibrillar collagen, and this shows that mechanical stability in skeletal muscle fibers, which is ensured by collagen IV, does not differ between ST and FT fibers. The effect of glucocorticoids on muscle weakness is applied through damaged contractile machinery of FT muscle fibers' intracellular compartment [23, 45].

2.2. Regeneration Capacity of Aging Myopathic Muscle. It has been shown that the turnover rate of contractile proteins in aging animals [49] and in young adults after the infusion of glucocorticoids decreases [23], and precursor cells required for muscle regrowth are morphologically and functionally damaged [38]. These changes together may be one of the reasons for sarcopenia (Figure 1). The mechanisms responsible for sarcopenia in aged skeletal muscle are largely unknown, but muscle satellite cells required for the repair of fibers certainly exhibit impaired activation [50] and proliferation [51] compared to young muscle.

Autografting of skeletal muscle has been used as a model of muscle regeneration. Higher oxidative capacity of muscle tends to ensure its faster regeneration [52–54]. It has been shown that the synthesis rate of contractile proteins depends on muscle oxidative potential [55]. In aging rats, the MyHC and actin synthesis rates decrease by about 30% and 23%, respectively [39]. It is known that aging is related to a dramatically reduced MyHC synthesis rate [56] without any change in MyHC on the transcriptional level [57]. Muscle wasting is also associated with increased protein degradation, particularly that of contractile proteins (Figure 1). Accumulation of abnormal proteins during aging is believed to result from defects in protein breakdown but very few experimental data support this hypothesis [22]. Results show that the degradation rate of contractile proteins in skeletal muscle during aging increased about two times, and dexamethasone treatment significantly increased the degradation rate in both age groups [39]. Previous works have shown that dexamethasone associated degradation starts from the periphery of myofibrils in muscle fibers with low oxidative potential [38]. This destruction process starts from myosin filaments and thereafter spreads all over the myofibrillar apparatus [38]. It has been shown that contractile proteins turned over slowly in old animals and subjects as well as in young rodents after dexamethasone treatment [34].

3. Effect of Unloading on Aging Skeletal Muscle

Aging is associated with a decline in skeletal muscle mass (sarcopenia), strength (dynapenia), and endurance (Figure 1). The term dynapenia was used to describe the age-related loss of muscle strength by Clark and Manini [58, 59]. Muscle unloading as a result of sedentary lifestyle, bed rest, spaceflight, and hindlimb suspension lead the skeletal muscle to microcirculatory disturbances, atrophy, protein loss, changes in contractile properties, and fiber-type switching [60].

The gradual development of functional limitations over an extended period of time is affected by the natural age-related decline in physical and biological properties, which already starts in midlife and increases the risk for a decline in physical functioning in later life [61].

During aging, the physical system suffers to a different extent and rate in diverse parts of the body. This results in reduced functional reserve, a decrease in vital capacity, deterioration of the capillary blood supply, and a decrease in muscle mass [62].

Living a sedentary life in older age, inactivity can lead to a loss of functional health due to deficits in muscle strength, endurance and flexibility [62]. "Use it or lose it" has proved to be a key rule for maintaining physical independence in the elderly [63]. One of the reasons for the development of muscle weakness in the elderly is decreased physical activity. Inactivity and aging cause a marked relative increase in the endo- and perimysial connective tissue, which results in changes in the mechanical properties of skeletal muscle [64].

Myofibrillar basal lamina becomes thicker and more rigid with age and increased cross-linking of collagen molecules make fibrils more resistant to degradation by collagenase [65]. The muscle tissue response to unloading seems to be more expressed than the connective tissue response [18, 66]. The connective structures are protected from rapid changes in tissue mass while muscle, which is known to act as a protein store for the organism, is subject to substantial and fast changes in tissue mass. Despite the small changes in connective tissue mass, important changes occur in the tissue structures during unloading and aging [4], which lead to the development of muscle weakness in case of restricted physical activity in the elderly.

4. The Preventive Role of Exercise Therapy on Aging Unloaded and Myopathic Muscle

Exercise therapy is a wide and systematic approach to the regular use of specific movements to improve different body functions, mobility, and fitness (Figure 2). Exercise therapy is a useful tool for the prevention and management of different injuries and diseases. On many occasions, specific exercise programs are tailored for rehabilitation needs. For example, in case of glucocorticoid caused myopathy, both endurance and strength exercise training has been shown to play a preventive role in the development of muscle atrophy, but a combination of both with different frequency, intensity and duration seems to be more effective (Figure 2).

More than four decades ago, the preventive role of exercise in the development of muscle atrophy during glucocorticoid administration was shown [25]. From the historical viewpoint, endurance exercise has been found to be an effective measure in retarding skeletal muscle atrophy associated with the administration of glucocorticoids [26, 29, 67]. From the contraction nature, four model systems have given the desired effect: endurance exercise, strength exercise, muscle functional overload, and in vitro cell culture stimulation [68]. Later intensive short-lasting exercise training has shown to have an anticatabolic effect on the contractile apparatus and the ECM of skeletal muscle [69]. Glucocorticoids increased myofibrillar protein degradation in FT muscles, while fibril- and network-forming collagen specific mRNA levels decreased at the same time in FT and ST muscles [45]. Both the myofibrillar apparatus and the ECM play a crucial role in changes of muscle strength during glucocorticoid administration and following muscle loading [70].

4.1. Effect of Resistance Exercise Training. Muscle atrophy contributes to but does not completely explain the decrease in force in the elderly. The age-related decrease in muscle mass and strength is a consequence of the complete loss of fibers associated with the decrease in the number of motor units and fiber atrophy [71]. In recent years, resistance exercise has become one of the fastest growing forms of physical activity for different purposes: improving athletic performance, enhancing general health and fitness, rehabilitation after surgery or an injury, or just for the pleasure of exercise [72]. Resistance exercise has shown to be an effective measure in the elderly, improving glucose intolerance, including improvements in insulin signaling defects, reduction in tumor necrosis factor-α, increases in adiponectin and IGF-1 concentrations, and reductions in total and abdominal visceral fat [73]. Resistance exercise improves skeletal muscle metabolism and through it muscle function in the elderly and their life quality [4].

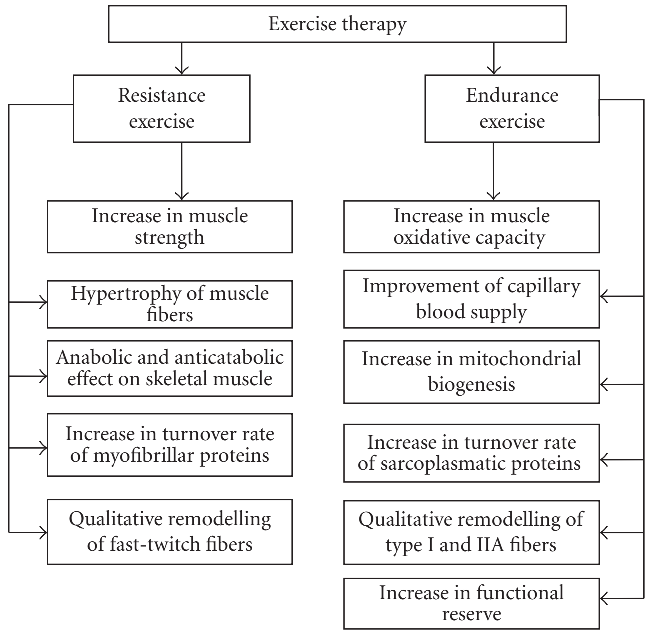


Figure 2: Effect of exercise therapy on aging, unloading, and glucocorticoid caused myopathic skeletal muscle.

Resistance exercise enhances the synthesis rate of myofibrillar proteins but not that of sarcoplasmic proteins [74] and this is related to mammalian target of rapamycin by activating proteins within the nitrogen-activated proteinkinase signaling [75]. A significant difference was observed between previously trained young and old participants in recovery from resistance training [76]. These results suggest a more rapid recovery in the young group. It seems that recovery from more damaging resistance exercise is slower as a result of age, whereas there are no age-related differences in recovery from less damaging metabolic fatigue [77].

It has been shown that resistance training, during which the power of exercise increased less than 5% per session, caused hypertrophy of both FT and ST muscle fibers, an increase of myonuclear number via fusion of satellite cells with damaged fibers or the formation of new muscle fibers as a result of myoblasts' fusion in order to maintain myonuclear domain size [78].

It has been shown that contractile proteins turned over faster in type I and IIA fibers than in IIB fibers and the turnover rate of skeletal muscle proteins in skeletal muscle depends on the functional activity of the muscle [30]. The turnover rate of myofibrillar proteins in aging skeletal muscle is related to the changes in MyHC isoforms' composition [4]. The effect of resistance training on the increase of the turnover rate of skeletal muscle contractile proteins in old age is relatively small [4]. Adaptational changes first appeared in newly formed or regenerating fibers and these changes lead to the remodeling of the contractile apparatus and an increase in the strength generating capability of muscle. These changes are more visible in muscle fibers with higher oxidative capacity. The recovery of locomotory activity after unloading is as fast as the recovery of muscle strength. It is related to the regeneration of muscle structure form disuse atrophy [79]. This fact suggests the presence of functionally immature muscle fibers during the recovery process following disuse atrophy [79]. So, the recovery of skeletal muscle mechanical properties depends on the structural and metabolic peculiarities of the skeletal muscle [30]. As a complex of factors contributes to the development of muscle wasting and weakness in the elderly, skeletal muscle unloading and glucocorticoid caused myopathy, it is complicated to find one certain measure for rehabilitation. As lack of strength is one of the central reasons for muscle weakness, it seems to be most realistic to use resistance training for this purpose in the elderly. Resistance training is a strong stimulus for muscle metabolism in the elderly, particularly for the contractile machinery of muscle (Figure 2).

4.2. Effect of Endurance Exercise Training. As oxidative capacity of skeletal muscle decreases in the elderly, endurance training is effective in stimulating mitochondrial biogenesis and improving their functional parameters [32, 33]. In combination with resistance training, the oxidative capacity and subsequently the turnover rate of contractile proteins in elderly skeletal muscle increases. This increase of the turnover rate of muscle proteins leads to the increase in skeletal muscle plasticity. It has recently been shown that the plasticity of individual development in the elderly makes it possible to modify the age-associated decline even in maximal physical performance [80]. Another positive influence of endurance training in the elderly is related to an increase in the ability of cardiovascular factors and to a lesser extent, to an increase in muscle mitochondrial concentration and capacity [81].

The increase in muscle oxidative capacity and contractile property is an effective measure for enhancing life quality in the elderly by improving skeletal muscle functional capacity and plasticity. It has recently been shown that the individual development of muscle plasticity in the elderly makes it possible to modify the age-associated decline even in maximal physical performance at least for some time [80]. The higher aerobic capacity in trained elderly people is related to an increase in the abilities of the cardiovascular system and to the lesser extent to an increase in muscle mitochondrial concentration [81]. It means that regular aerobic activity provides a foundation for an increase in muscle oxidative capacity in the elderly (Figure 2). It is useful to repeat the viewpoint of Suominen [80] that adequate physical performance is an essential element of a healthy and productive life among the elderly. Netz [82] studied the effect of physical activity on the moderating role of fitness improvement and mode of exercise to the potential mechanisms for explaining the physical activity affect relationship and found that neither improved fitness nor exercise modality serve as moderators of physical activity effect on affect. However, with older age managing everyday activities becomes less self-evident although there are gender differences in physical functioning [83]. Functional limitation is an objective measure of the consequences of disease and impairment [84].

It seems that the turnover rate of contractile proteins provides a mechanism by which the effect of exercise causes changes in accordance with the needs of the contractile apparatus. As the contractile protein turnover rate depends on the oxidative capacity of muscle and muscle oxidative capacity decreases in the elderly, it is obvious that endurance exercise stimulates an increase in the oxidative capacity of skeletal muscle by an increase in mitochondrial biogeneses and supports faster protein turnover during resistance training in order to increase muscle function (Figure 2). It has been shown that the aging-associated reduction in AMP-activated protein kinase (AMPK) activity may be a factor in reduced mitochondrial function [85]. In response to contractile activity, AMPK activation was registered only in aging FT muscles [86]. It is known that AMPK is activated in response to endurance exercise [87] and related to the metabolic adaptation of skeletal muscle. Later it has been shown that $\alpha 1$ isoform of AMPK is the regulator of skeletal muscle growth, but not of metabolic adaptation [66]. As factors such as health, physical function, and independence constitute components of quality of life in the elderly, physiological functioning of skeletal muscle in the elderly has significance in determining the ability to maintain independence and an active interaction with the environment [4, 88]. According to Kramer and Erickson [89], successful aging is guaranteed when elderly people use widespread participation in low-cost and low-tech exercise for further improving their fitness and reducing the risk of disability.

4.3. Effect of Concurrent Strength and Endurance Exercise Training. Concurrent training for strength and endurance has shown to decrease the gain in muscle mass in comparison with training for strength alone [90]. This effect was explained by AMPK blocking the activation of mammalian target of rapamycin complex-1 (TORC 1) by phosphorylating and activating the tuberous sclerosis complex-2 (TSC 2) [91]. This interference in skeletal muscle strength development was also explained by alterations in the protein synthesis induced by the high volume of endurance exercise or by frequent exercise training sessions [92] or was related to impairment of neural adaptations [93].

Concurrent strength and endurance exercise training in elderly men has shown that strength gain was similar to that observed with strength training alone, although strength training volume was half of that strength training alone [94]. Using lower training volumes in concurrent training in older men [95] in comparison with endurance and resistance training alone leads to similar strength enhancement with no presence of interference in this population [96]. In the elderly population, improvement in both strength and cardiorespiratory fitness is important and concurrent training is the best strategy to enhance cardiorespiratory fitness as it has widely been shown in the literature [93].

5. Conclusions and Future Directions

Changes in skeletal muscle mass and function with advancing age are reasons for disability in the aging population. Glucocorticoid treatment and skeletal muscle unloading lead to muscle atrophy, loss of myofibrillar proteins, changes in ECM and a decrease in muscle strength and motor activity. As a complex of factors supporting development of skeletal muscle wasting and weakness in the elderly in case of muscle unloading and glucocorticoid caused myopathy it is complicated to find one certain facility for rehabilitation. As the lack of strength is one of the central reasons in muscle weakness, it seems to be promising to use resistance training for this purpose in elderly. On the other hand, as oxidative capacity of skeletal muscle decreases in the elderly and endurance training is known to be an effective tool in the stimulation of mitochondrial biogenesis and improving their functional parameters, it seems that in combination with resistance exercise training, the oxidative capacity and subsequently the turnover rate of muscle contractile proteins in elderly skeletal muscle increases and leads to an increase in muscle plasticity. Physiological functioning of skeletal muscle in the elderly has significance in determining the ability to maintain independence and an active interaction with the environment.

Aging-associated reductions in AMPK activity may be a factor in the reduced mitochondrial function and AMPK is activated in response to endurance exercise, which explains the use of endurance exercise training in the prevention of disability and diseases. Frailty due to sarcopenia and dynapenia are proved reasons for the loss of an active interaction with the environment and loss of independence in the elderly, and predicate the use of resistance exercise for purposes of increasing muscle strength. The use of concurrent strength and endurance training in the prevention of muscle atrophy in the elderly population and the connection with anabolic and anticatabolic processes in skeletal muscle has not yet been elucidated. Future studies should focus on concurrent strength and endurance exercise effects on the prevention of skeletal muscle atrophy in the elderly during unloading and glucocorticoid treatment. The question is whether and in what conditions AMPK blocks the activation of TORC 1 by activating the TSC 2 during concurrent strength and endurance exercise in elderly skeletal muscle. To the best of our knowledge, it seems that concurrent training is an auspicious tool in the prevention or at least in the deceleration of the development of sarcopenia and dynapenia in the elderly. The important step is to optimize exercise therapy programs with respect to the interaction between signaling pathways for contractile and mitochondrial protein synthesis and degradation in the aging unloaded and myopathic population. This strategy to work out concurrent strength and endurance exercise programs in the elderly is complicated as it is unclear whether a muscle fiber is capable to undergo hypertrophy and maintain endurance capacity at the same time. It is clear that an exercise program has to be composed, which enables the recruitment of both ST and FT muscle fibers for the purpose of contributing the signaling pathways to accelerate protein turnover in aging skeletal muscle.

Acknowledgments

This study was supported by the funds of the Ministry of Education and Research of the Republic of Estonia, Research Project no. TKKSB1787. The authors would like to thank Helen Kaptein for English language editing and Piret Pärsim for technical expertise.

References

[1] J. M. Haus, J. A. Carrithers, S. W. Trappe, and T. A. Trappe, "Collagen, cross-linking, and advanced glycation end products in aging human skeletal muscle," *Journal of Applied Physiology*, vol. 103, no. 6, pp. 2068–2076, 2007.

[2] T. Trappe, "Influence of aging and long-term unloading on the structure and function of human skeletal muscle," *Applied Physiology, Nutrition and Metabolism*, vol. 34, no. 3, pp. 459–464, 2009.

[3] C. A. D. S. Santos, E. E. M. Dantas, and M. H. R. Moreira, "Correlation of physical aptitude; functional capacity, corporal balance and quality of life (QoL) among elderly women submitted to a post-menopausal physical activities program," *Archives of Gerontology and Geriatrics*, vol. 53, pp. 344–349, 2011.

[4] T. Seene, P. Kaasik, and E. M. Riso, "Review on aging, unloading and reloading: changes in skeletal muscle quantity and quality," *Archives of Gerontology and Geriatrics*, vol. 54, no. 2, pp. 374–280, 2011.

[5] E. Marzetti and C. Leeuwenburgh, "Skeletal muscle apoptosis, sarcopenia and frailty at old age," *Experimental Gerontology*, vol. 41, no. 12, pp. 1234–1238, 2006.

[6] A. Hiona and C. Leeuwenburgh, "The role of mitochondrial DNA mutations in aging and sarcopenia: implications for the mitochondrial vicious cycle theory of aging," *Experimental Gerontology*, vol. 43, no. 1, pp. 24–33, 2008.

[7] P. M. Siu, E. E. Pistilli, and S. E. Alway, "Age-dependent increase in oxidative stress in gastrocnemius muscle with unloading," *Journal of Applied Physiology*, vol. 105, no. 6, pp. 1695–1705, 2008.

[8] S. K. Powers, A. N. Kavazis, and J. M. McClung, "Oxidative stress and disuse muscle atrophy," *Journal of Applied Physiology*, vol. 102, no. 6, pp. 2389–2397, 2007.

[9] N. Luden, K. Minchev, E. Hayes, E. Louis, T. Trappe, and S. Trappe, "Human vastus lateralis and soleus muscles display divergent cellular contractile properties," *American Journal of Physiology*, vol. 295, no. 5, pp. R1593–R1598, 2008.

[10] C. Leeuwenburgh, C. M. Gurley, B. A. Strotman, and E. E. Dupont-Versteegden, "Age-related differences in apoptosis with disuse atrophy in soleus muscle," *American Journal of Physiology*, vol. 288, no. 5, pp. R1288–R1296, 2005.

[11] A. S. Sreedhar and P. Csermely, "Heat shock proteins in the regulation of apoptosis: new strategies in tumor therapy: a comprehensive review," *Pharmacology and Therapeutics*, vol. 101, no. 3, pp. 227–257, 2004.

[12] T. Ogata, S. Machida, Y. Oishi, M. Higuchi, and I. Muraoka, "Differential cell death regulation between adult-unloaded

and aged rat soleus muscle," *Mechanisms of Ageing and Development*, vol. 130, no. 5, pp. 328–336, 2009.

[13] J. H. Kim, H. B. Kwak, C. Leeuwenburgh, and J. M. Lawler, "Lifelong exercise and mild (8%) caloric restriction attenuate age-induced alterations in plantaris muscle morphology, oxidative stress and IGF-1 in the fischer-344 rat," *Experimental Gerontology*, vol. 43, no. 4, pp. 317–329, 2008.

[14] T. W. Buford, S. D. Anton, A. R. Judge et al., "Models of accelerated sarcopenia: critical pieces for solving the puzzle of age-related muscle atrophy," *Ageing Research Reviews*, vol. 9, no. 4, pp. 369–383, 2010.

[15] R. Roubenoff, "Catabolism of aging: is it an inflammatory process?" *Current Opinion in Clinical Nutrition and Metabolic Care*, vol. 6, no. 3, pp. 295–299, 2003.

[16] G. Goldspink and S. D. R. Harridge, "Growth factors and muscle ageing," *Experimental Gerontology*, vol. 39, no. 10, pp. 1433–1438, 2004.

[17] M. J. Toth and A. Tchernof, "Lipid metabolism in the elderly," *European Journal of Clinical Nutrition*, vol. 54, supplement 3, pp. S121–S125, 2000.

[18] M. Kjær, P. Magnusson, M. Krogsgaard et al., "Extracellular matrix adaptation of tendon and skeletal muscle to exercise," *Journal of Anatomy*, vol. 208, no. 4, pp. 445–450, 2006.

[19] K. M. Heinemeier, J. L. Olesen, F. Haddad, P. Schjerling, K. M. Baldwin, and M. Kjaer, "Effect of unloading followed by reloading on expression of collagen and related growth factors in rat tendon and muscle," *Journal of Applied Physiology*, vol. 106, no. 1, pp. 178–186, 2009.

[20] A. J. Freemont and J. A. Hoyland, "Morphology, mechanisms and pathology of musculoskeletal ageing," *Journal of Pathology*, vol. 211, no. 2, pp. 252–259, 2007.

[21] D. Pottle and L. E. Gosselin, "Impact of mechanical load on functional recovery after muscle reloading," *Medicine and Science in Sports and Exercise*, vol. 32, no. 12, pp. 2012–2017, 2000.

[22] D. Attaix, L. Mosoni, D. Dardevet, L. Combaret, P. P. Mirand, and J. Grizard, "Altered responses in skeletal muscle protein turnover during aging in anabolic and catabolic periods," *International Journal of Biochemistry and Cell Biology*, vol. 37, no. 10, pp. 1962–1973, 2005.

[23] T. Seene, P. Kaasik, A. Pehme, K. Alev, and E. M. Riso, "The effect of glucocorticoids on the myosin heavy chain isoforms' turnover in skeletal muscle," *Journal of Steroid Biochemistry and Molecular Biology*, vol. 86, no. 2, pp. 201–206, 2003.

[24] W. Evans, "Functional and metabolic consequences of sarcopenia," *Journal of Nutrition*, vol. 127, no. 5, pp. 9985–10035, 1997.

[25] A. L. Goldberg and H. M. Goodman, "Relationship between cortisone and muscle work in determining muscle size," *Journal of Physiology*, vol. 200, no. 3, pp. 667–675, 1969.

[26] T. Seene and A. Viru, "The catabolic effect of glucocorticoids on different types of skeletal muscle fibres and its dependence upon muscle activity and interaction with anabolic steroids," *Journal of Steroid Biochemistry*, vol. 16, no. 2, pp. 349–352, 1982.

[27] R. C. Hickson and J. R. Davis, "Partial prevention of glucocorticoid-induced muscle atrophy by endurance training," *The American Journal of Physiology*, vol. 241, no. 3, pp. E226–E232, 1981.

[28] R. C. Hickson, T. M. Galassi, J. A. Capaccio, and R. T. Chatterton, "Limited resistance of hypertrophied skeletal muscle to glucocorticoids," *Journal of Steroid Biochemistry*, vol. 24, no. 6, pp. 1179–1183, 1986.

[29] S. M. Czerwinski-Helms and R. C. Hickson, "Specificity of activated glucocorticoid receptor expression in heart and skeletal muscle types," *Biochemical and Biophysical Research Communications*, vol. 142, no. 2, pp. 322–328, 1987.

[30] T. Seene, P. Kaasik, and M. Umnova, "Structural rearrangements in contractile apparatus and resulting skeletal muscle remodelling: effect of exercise training," *Journal of Sports Medicine and Physical Fitness*, vol. 49, no. 4, pp. 410–423, 2009.

[31] T. Seene, P. Kaasik, and K. Alev, "Muscle protein turnover in endurance training: a review," *International Journal of Sports Medicine*, vol. 32, no. 12, pp. 905–911, 2011.

[32] D. A. Hood, "Mechanisms of exercise-induced mitochondrial biogenesis in skeletal muscle," *Applied Physiology, Nutrition and Metabolism*, vol. 34, no. 3, pp. 465–472, 2009.

[33] V. Ljubicic, A. M. Joseph, A. Saleem et al., "Transcriptional and post-transcriptional regulation of mitochondrial biogenesis in skeletal muscle: effects of exercise and aging," *Biochimica et Biophysica Acta*, vol. 1800, no. 3, pp. 223–234, 2010.

[34] T. Seene, "Turnover of skeletal muscle contractile proteins in glucocorticoid myopathy," *Journal of Steroid Biochemistry and Molecular Biology*, vol. 50, no. 1-2, pp. 1–4, 1994.

[35] D. Dardevet, C. Sornet, D. Taillandier, I. Savary, D. Attaix, and J. Grizard, "Sensitivity and protein turnover response to glucocorticoids are different in skeletal muscle from adult and old rats. Lack of regulation of the ubiquitin-proteasome proteolytic pathway in aging," *Journal of Clinical Investigation*, vol. 96, no. 5, pp. 2113–2119, 1995.

[36] D. Dardevet, C. Sornet, I. Savary, E. Debras, P. Patureau-Mirand, and J. Grizard, "Glucocorticoid effects on insulin- and IGF-I-regulated muscle protein metabolism during aging," *Journal of Endocrinology*, vol. 156, no. 1, pp. 83–89, 1998.

[37] J. N. Artaza, S. Bhasin, C. Mallidis, W. Taylor, K. Ma, and N. F. Gonzalez-Cadavid, "Endogenous expression and localization of myostatin and its relation to myosin heavy chain distribution in C2C12 skeletal muscle cells," *Journal of Cellular Physiology*, vol. 190, no. 2, pp. 170–179, 2002.

[38] T. Seene, M. Umnova, K. Alev, and A. Pehme, "Effect of glucocorticoids on contractile apparatus of rat skeletal muscle," *Journal of Steroid Biochemistry*, vol. 29, no. 3, pp. 313–317, 1988.

[39] P. Kaasik, M. Umnova, A. Pehme et al., "Ageing and dexamethasone associated sarcopenia: peculiarities of regeneration," *Journal of Steroid Biochemistry and Molecular Biology*, vol. 105, no. 1–5, pp. 85–90, 2007.

[40] K. R. Short and K. S. Nair, "The effect of age on protein metabolism," *Current Opinion in Clinical Nutrition and Metabolic Care*, vol. 3, no. 1, pp. 39–44, 2000.

[41] J. Järva, K. Alev, and T. Seene, "Myosin heavy chain composition in regenerating skeletal muscle grafts," *Basic and Applied Myology*, vol. 7, no. 2, pp. 137–141, 1997.

[42] J. Järva, K. Alev, and T. Seene, "The effect of autografting on the myosin composition in skeletal muscle fibers," *Muscle & Nerve*, vol. 20, no. 6, pp. 718–727, 1997.

[43] T. Seene, M. Umnova, P. Kaasik, K. Alev, and A. Pehme, "Overtraining injuries in athletic population," in *Skeletal Muscle Damage and Repair*, P. Tiidus, Ed., Human Kinetics Books, Champaign, Ill, USA, 2008.

[44] A. M. Ahtikoski, E. M. Riso, S. O. A. Koskinen, J. Risteli, and T. E. S. Takala, "Regulation of type IV collagen gene expression and degradation in fast and slow muscles during dexamethasone treatment and exercise," *Pflugers Archiv*, vol. 448, no. 1, pp. 123–130, 2004.

[45] E. M. Riso, A. Ahtikoski, K. Alev, P. Kaasik, A. Pehme, and T. Seene, "Relationship between extracellular matrix, contractile apparatus, muscle mass and strength in case of glucocorticoid myopathy," *Journal of Steroid Biochemistry and Molecular Biology*, vol. 108, no. 1-2, pp. 117–120, 2008.

[46] V. Everts, E. van der Zee, L. Creemers, and W. Beertsen, "Phagocytosis and intracellular digestion of collagen, its role in turnover and remodelling," *Histochemical Journal*, vol. 28, no. 4, pp. 229–245, 1996.

[47] V. Kovanen, H. Suominen, and E. Heikkinen, "Mechanical properties of fast and slow skeletal muscle with special reference to collagen and endurance training," *Journal of Biomechanics*, vol. 17, no. 10, pp. 725–735, 1984.

[48] V. Kovanen, H. Suominen, and E. Heikkinen, "Collagen of slow twitch and fast twitch muscle fibres in different types of rat skeletal muscle," *European Journal of Applied Physiology and Occupational Physiology*, vol. 52, no. 2, pp. 235–242, 1984.

[49] A. Pehme, K. Alev, P. Kaasik, and T. Seene, "Age-related changes in skeletal-muscle myosin heavy-chain composition: effect of mechanical loading," *Journal of Aging and Physical Activity*, vol. 12, no. 1, pp. 29–44, 2004.

[50] A. E. Barani, A. C. Durieux, O. Sabido, and D. Freyssenet, "Age-related changes in the mitotic and metabolic characteristics of muscle-derived cells," *Journal of Applied Physiology*, vol. 95, no. 5, pp. 2089–2098, 2003.

[51] S. Machida and F. W. Booth, "Increased nuclear proteins in muscle satellite cells in aged animals as compared to young growing animals," *Experimental Gerontology*, vol. 39, no. 10, pp. 1521–1525, 2004.

[52] B. M. Carlson, "Regeneration of entire skeletal muscles," *Federation Proceedings*, vol. 45, no. 5, pp. 1456–1460, 1986.

[53] E. Shultz and K. Darr, "The role of satellite cells in adaptive or induced fiber transformations," in *The Dynamic State of Muscle Fibers*, D. Pette, Ed., pp. 667–681, Walter De Gruyter, Berlin, Germany, 1990.

[54] T. P. White and S. T. Devor, "Skeletal muscle regeneration and plasticity of grafts," *Exercise and Sport Sciences Reviews*, vol. 21, pp. 263–295, 1993.

[55] T. Seene, P. Kaasik, K. Alev, A. Pehme, and E. M. Riso, "Composition and turnover of contractile proteins in volume-overtrained skeletal muscle," *International Journal of Sports Medicine*, vol. 25, no. 6, pp. 438–445, 2004.

[56] P. Balagopal, O. E. Rooyackers, D. B. Adey, P. A. Ades, and K. S. Nair, "Effects of aging on in vivo synthesis of skeletal muscle myosin heavy- chain and sarcoplasmic protein in humans," *American Journal of Physiology*, vol. 273, no. 4, pp. E790–E800, 1997.

[57] S. Welle, K. Bhatt, and C. Thornton, "Polyadenylated RNA, actin mRNA, and myosin heavy chain mRNA in young and old human skeletal muscle," *American Journal of Physiology*, vol. 270, no. 2, pp. E224–E229, 1996.

[58] B. C. Clark and T. M. Manini, "Sarcopenia ≠ dynapenia," *Journals of Gerontology A*, vol. 63, no. 8, pp. 829–834, 2008.

[59] T. M. Manini and B. C. Clark, "Dynapenia and aging: an uptate," *Journal of Gerontology*, vol. 67A, no. 1, pp. 28–40, 2012.

[60] T. Trappe, "Influence of aging and long-term unloading on the structure and function of human skeletal muscle," *Applied Physiology, Nutrition and Metabolism*, vol. 34, no. 3, pp. 459–464, 2009.

[61] M. B. von Bonsdorff and T. Rantanen, "Progression of functional limitations in relation to physical activity: a life course approach," *European Review of Aging and Physical Activity*, vol. 8, no. 1, pp. 23–30, 2011.

[62] H. Mechling and Y. Netz, "Aging and inactivity-capitalizing on the protective effect of planned physical activity in old age," *European Review of Aging and Physical Activity*, vol. 6, no. 2, pp. 89–97, 2009.

[63] W. J. Rejeski and L. R. Brawley, "Functional health: innovations in research on physical activity with older adults," *Medicine and Science in Sports and Exercise*, vol. 38, no. 1, pp. 93–99, 2006.

[64] C. Ducomps, P. Mauriège, B. Darche, S. Combes, F. Lebas, and J. P. Doutreloux, "Effects of jump training on passive mechanical stress and stiffness in rabbit skeletal muscle: role of collagen," *Acta Physiologica Scandinavica*, vol. 178, no. 3, pp. 215–224, 2003.

[65] G. Goldspink, K. Fernandes, P. E. Williams, and D. J. Wells, "Age-related changes in collagen gene expression in the muscles of mdx dystrophic and normal mice," *Neuromuscular Disorders*, vol. 4, no. 3, pp. 183–191, 1994.

[66] S. L. Mcgee, K. J. Mustard, D. G. Hardie, and K. Baar, "Normal hypertrophy accompanied by phosphoryation and activation of AMP-activated protein kinase α1 following overload in LKB1 knockout mice," *Journal of Physiology*, vol. 586, no. 6, pp. 1731–1741, 2008.

[67] R. C. Hickson, T. T. Kurowski, J. A. Capaccio, and R. T. Chatterton, "Androgen cytosol binding in exercise-induced sparing of muscle atrophy," *American Journal of Physiology*, vol. 247, no. 5, part 1, pp. E597–E603, 1984.

[68] S. M. Czerwinski and R. C. Hickson, "Glucocorticoid receptor activation during exercise in muscle," *Journal of Applied Physiology*, vol. 68, no. 4, pp. 1615–1620, 1990.

[69] E.-M. Riso, A. M. Ahtikoski, M. Umnova et al., "Partial prevention of muscle atrophy in excessive level of glucocorticoids by exercise: effect on contractile proteins and extracellular matrix," *Baltic Journal of Laboratory Animal Science*, vol. 13, no. 1, pp. 5–12, 2003.

[70] E. M. Riso, A. M. Ahtikoski, T. E. S. Takala, and T. Seene, "The effect of unloading and reloading on the extracellular matrix in skeletal muscle: changes in muscle strength and motor activity," *Biology of Sport*, vol. 27, no. 2, pp. 89–94, 2010.

[71] E. P. Rader and J. A. Faulkner, "Recovery from contraction-induced injury is impaired in weight-bearing muscles of old male mice," *Journal of Applied Physiology*, vol. 100, no. 2, pp. 656–661, 2006.

[72] A. C. Fry, "The role of resistance exercise intensity on muscle fibre adaptations," *Sports Medicine*, vol. 34, no. 10, pp. 663–679, 2004.

[73] K. D. Flack, K. P. Davy, M. W. Hulver et al., "Aging, resistance training, and diabetes prevention," *Journal of Aging Research*, vol. 2011, Article ID 127315, 12 pages, 2011.

[74] D. R. Moore, J. E. Tang, N. A. Burd, T. Rerecich, M. A. Tarnopolsky, and S. M. Phillips, "Differential stimulation of myofibrillar and sarcoplasmic protein synthesis with protein ingestion at rest and after resistance exercise," *Journal of Physiology*, vol. 587, no. 4, pp. 897–904, 2009.

[75] D. R. Moore, P. J. Atherton, M. J. Rennie, M. A. Tarnopolsky, and S. M. Phillips, "Resistance exercise enhances mTOR and MAPK signalling in human muscle over that seen at rest after bolus protein ingestion," *Acta Physiologica*, vol. 201, no. 3, pp. 365–372, 2011.

[76] J. R. McLester, P. A. Bishop, J. Smith et al., "A series of studies—a practical protocol for testing muscular endurance recovery," *Journal of Strength and Conditioning Research*, vol. 17, no. 2, pp. 259–273, 2003.

[77] J. Fell and A. D. Williams, "The effect of aging on skeletal-muscle recovery from exercise: possible implications for aging

athletes," *Journal of Aging and Physical Activity*, vol. 16, no. 1, pp. 97–115, 2008.

[78] T. Seene, A. Pehme, K. Alev, P. Kaasik, M. Umnova, and M. Aru, "Effects of resistance training on fast- and slow-twitch muscles in rats," *Biology of Sport*, vol. 27, no. 3, pp. 221–229, 2010.

[79] Y. Itai, Y. Kariya, and Y. Hoshino, "Morphological changes in rat hindlimb muscle fibres during recovery from disuse atrophy," *Acta Physiologica Scandinavica*, vol. 181, no. 2, pp. 217–224, 2004.

[80] H. Suominen, "Ageing and maximal physical performance," *European Review of Aging and Physical Activity*, vol. 8, no. 1, pp. 37–42, 2011.

[81] M. Sagiv, E. Goldhammer, D. Ben-Sira, and R. Amir, "Factors defining oxygen uptake at peak exercise in aged people," *European Review of Aging and Physical Activity*, vol. 7, no. 1, pp. 1–2, 2010.

[82] Y. Netz, "Type of activity and fitness benefits as moderators of the effect of physical activity on affect in advanced age: a review," *European Review of Aging and Physical Activity*, vol. 6, no. 1, pp. 19–27, 2009.

[83] D. Kuh, E. J. Bassey, S. Butterworth, R. Hardy, and M. E. J. Wadsworth, "Grip strength, postural control, and functional leg power in a representative cohort of British men and women: associations with physical activity, health status, and socioeconomic conditions," *Journals of Gerontology A*, vol. 60, no. 2, pp. 224–231, 2005.

[84] J. M. Guralnik and L. Ferrucci, "Assessing the building blocks of function: utilizing measures of functional limitation," *American Journal of Preventive Medicine*, vol. 25, no. 3, pp. 112–121, 2003.

[85] R. M. Reznick, H. Zong, J. Li et al., "Aging-associated reductions in AMP-activated protein kinase activity and mitochondrial biogenesis," *Cell Metabolism*, vol. 5, no. 2, pp. 151–156, 2007.

[86] D. M. Thomson, J. D. Brown, N. Fillmore et al., "AMP-activated protein kinase response to contractions and treatment with the AMPK activator AICAR in young adult and old skeletal muscle," *Journal of Physiology*, vol. 587, no. 9, pp. 2077–2086, 2009.

[87] W. W. Winder and D. G. Hardie, "Inactivation of acetyl-CoA carboxylase and activation of AMP-activated protein kinase in muscle during exercise," *American Journal of Physiology*, vol. 270, no. 2, pp. E299–E304, 1996.

[88] W. W. Spirduso and D. L. Cronin, "Exercise dose-response effects on quality of life and independent living in older adults," *Medicine and Science in Sports and Exercise*, vol. 33, supplement 6, pp. S598–S608, 2001.

[89] A. F. Kramer and K. I. Erickson, "Capitalizing on cortical plasticity: influence of physical activity on cognition and brain function," *Trends in Cognitive Sciences*, vol. 11, no. 8, pp. 342–348, 2007.

[90] R. C. Hickson, "Interference of strength development by simultaneously training for strength and endurance," *European Journal of Applied Physiology and Occupational Physiology*, vol. 45, no. 2-3, pp. 255–263, 1980.

[91] K. Inoki, Y. Li, T. Zhu, J. Wu, and K. L. Guan, "TSC2 is phosphorylated and inhibited by Akt and suppresses mTOR signalling," *Nature Cell Biology*, vol. 4, no. 9, pp. 648–657, 2002.

[92] G. A. Nader, "Concurrent strength and endurance training: from molecules to man," *Medicine and Science in Sports and Exercise*, vol. 38, no. 11, pp. 1965–1970, 2006.

[93] E. L. Cadore, R. S. Pinto, F. L. R. Lhullier et al., "Physiological effects of concurrent training in elderly men," *International Journal of Sports Medicine*, vol. 31, no. 10, pp. 689–697, 2010.

[94] R. H. Wood, R. Reyes, M. A. Welsch et al., "Concurrent cardiovascular and resistance training in healthy older adults," *Medicine and Science in Sports and Exercise*, vol. 33, no. 10, pp. 1751–1758, 2001.

[95] M. Izquierdo, J. Ibañez, K. Häkkinen, W. J. Kraemer, J. L. Larrión, and E. M. Gorostiaga, "Once weekly combined resistance and cardiovascular training in healthy older men," *Medicine and Science in Sports and Exercise*, vol. 36, no. 3, pp. 435–443, 2004.

[96] L. Karavirta, M. P. Tulppo, D. E. Laaksonen et al., "Heart rate dynamics after combined endurance and strength training in older men," *Medicine and Science in Sports and Exercise*, vol. 41, no. 7, pp. 1436–1443, 2009.

Hindawi Publishing Corporation
Journal of Aging Research
Volume 2012, Article ID 950192, 7 pages
doi:10.1155/2012/950192

Review Article

Aging and Osteoarthritis: An Inevitable Encounter?

Thomas Hügle,[1,2] Jeroen Geurts,[1] Corina Nüesch,[1]
Magdalena Müller-Gerbl,[3] and Victor Valderrabano[1]

[1] *Osteoarthritis Research Center, Department of Orthopaedic Surgery, University Hospital Basel, Basel University, Spitalstrasse 21, 4031 Basel, Switzerland*
[2] *Department of Rheumatology, University Hospital Basel, Felix-Platter-Spital, 4012 Basel, Switzerland*
[3] *Institute of Anatomy, University of Basel, 4056 Basel, Switzerland*

Correspondence should be addressed to Thomas Hügle, thomas.huegle@unibas.ch

Received 24 February 2012; Accepted 28 March 2012

Academic Editor: Iris Reuter

Osteoarthritis (OA) is a major health burden of our time. Age is the most prominent risk factor for the development and progression of OA. The mechanistic influence of aging on OA has different facets. On a molecular level, matrix proteins such as collagen or proteoglycans are modified, which alters cartilage function. Collagen cross-linking within the bone results in impaired plasticity and increased stiffness. Synovial or fat tissue, menisci but also ligaments and muscles play an important role in the pathogenesis of OA. In the elderly, sarcopenia or other causes of muscle atrophy are frequently encountered, leading to a decreased stability of the joint. Inflammation in form of cellular infiltration of synovial tissue or subchondral bone and expression of inflammatory cytokines is more and more recognized as trigger of OA. It has been demonstrated that joint movement can exhibit anti-inflammatory mechanisms. Therefore physical activity or physiotherapy in the elderly should be encouraged, also in order to increase the muscle mass. A reduced stem cell capacity in the elderly is likely associated with a decrease of repair mechanisms of the musculoskeletal system. New treatment strategies, for example with mesenchymal stem cells (MSC) are investigated, despite clear evidence for their efficacy is lacking.

1. Introduction

Half of all persons aged over 65 suffer from osteoarthritis (OA) [1]. As a matter of fact, age is the most prominent risk factor for the initiation and progression of OA. The common explanation for this is the cumulative effect of mechanical load over the years, resulting clinically in "wear and tear" and pathologically in cartilage breakdown [2]. Therefore, OA has been regarded as a naturally occurring, irreversible disorder, rather than a specific, potentially treatable disease. During the last decade, however, it became clearer that OA is not a purely mechanical problem. Inflammatory and metabolic processes are substantially involved in the pathogenesis and progression of OA. Not only cartilage, but also subchondral bone, menisci, muscles as well as fat, and synovial tissues play an important role, notably in the early phase of OA (Figure 1). Therefore, OA has been referred to as a "whole joint disease." Despite a higher complexity, this concept has not only improved our understanding of the disease but also indicates potentially new treatment strategies.

To understand why aging predisposes to the development of OA, a link between aging processes and the pathological changes in the OA joint needs to be established. On a molecular level, aging research has revealed intrinsic changes in the structure of extracellular matrix proteins such as collagen or proteoglycans. Stiffening of the collagen network or increased glycation provoke a functional impairment of cartilage and joint function [3]. Aging also has profound effects on cellular processes notably leading to enhanced apoptosis and reduced cellular regeneration [4].

Nonenzymatic collagen cross-linking leads abnormalities in bone toughness and stiffness. Bone plasticity is further suppressed by an increase of osteon density, which leads to a lower potency of crack-bridging mechanisms [5].

Synovitis is frequently involved in OA, notably in the early phase of the disease [6]. This has been demonstrated both by histological studies and magnetic resonance imaging (MRI) analyses [7]. OA synovial fluid contains proinflammatory cytokines. This is demonstrated clinically when a ruptured baker cyst leads to painful swelling of

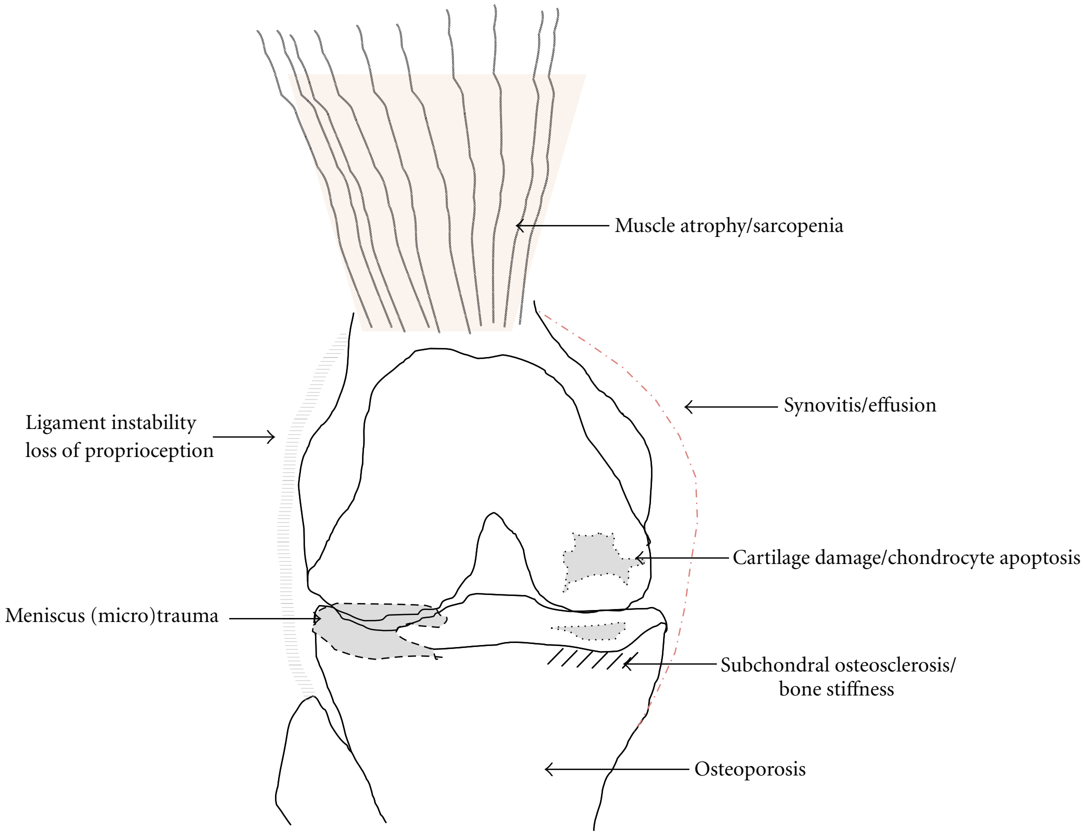


Figure 1: Osteoarthritis as a whole joint disease in the elderly.

the surrounding soft tissue. Similarly, the penetration of synovial fluid into the subchondral space after microfracture of the subchondral bone plate can induce an inflammatory response.

The exact reason for the inflamed status of the OA joint remains unclear. Overuse can lead to an activation of osteoblasts but also mast cells. Subsequently, other immune cells of the innate or adaptive immune system are attracted. Crystals, for example, in calcium pyrophosphate disease, a typical bystander in OA can activate the inflammasome leading to interleukin-1 activation [8]. On the other hand, during aging the immune system is less capable to resolve inflammation in general. In other words, not only the initiation of inflammation, but also the lack of inflammation might be involved in OA, notably in the aged individual.

Notwithstanding, mechanical dysfunction is still regarded as key player in OA pathogenesis, predominantly in weight bearing joints. This is also referred as "continuous loading theory." Apart from the aforementioned molecular impairment of cartilage, also the musculature is involved in stabilization and therefore protection of the joint. Muscle atrophy is well recognized in OA pathogenesis [9]. In the elderly, muscle atrophy is highly prevalent; this can be caused by sarcopenia in general, lack of physical training, malnutrition or arthrogenic muscle inhibition as a direct consequence of OA.

Finally, aging leads to a reduced regenerative capacity, for example, shown by the reduced levels of stem cells in connective tissue in the elderly. The elderly enters a catabolic state, where a presenescent cell state leads to subsequent loss of connective tissue homeostasis.

In this paper we give an overview of age-related structural and functional changes occurring in the course of OA.

2. Changes in Gait Biomechanics in OA

Changes in the joint biomechanics of patients suffering from OA have been shown in various studies. Gait analyses showed that patients with OA in the medial compartment of the knee walk with a more extended knee at heel strike [10]. Additionally, the external knee adduction moment has been identified as an important factor in medial knee OA [10–12]. The peak knee adduction moment has been shown to increase with increasing varus malalignment [12] and with increasing radiographic disease severity [10, 11]. Although the knee adduction moment has been identified as a risk factor for disease progression [11], there is also evidence that in early stages of OA, patients are able to reduce the knee adduction moment by moving the trunk laterally [10]. However, to achieve this, sufficient hip abduction muscle strength is needed to balance the external hip adduction moment.

In a recent meta-analysis, Pietrosimone et al. [13] showed that patients with knee OA have a deficit in the activation of the quadriceps muscles. Additionally, there is evidence that weak knee extensor increases the risk to develop symptomatic knee OA [14, 15]. The muscles surrounding the knee joint are important for the stability. Muscle weakness can therefore impair the neuromuscular protection of the joint leading to microtraumas and possibly joint damage [16]. Hence, muscular dysfunction might not only be a consequence of the disease but could also play a role in the development of OA [14, 16].

3. Biomechanical Changes in the Elderly

Walking speed is relatively consistent between age 20 and 70 and declines after age 70 [17, 18]. Older people often walk with a reduced step length which is a possible adaptation to lower muscle strength and fear of tripping and falling [19, 20]. In this study it has been shown that patients with knee OA produce lower knee extension forces than age-matched controls. These force levels were comparable to those of an on average 20 years older age group. In contrast to the knee OA patients that also showed joint laxity, the healthy elderly could maintain normal joint biomechanics during walking by increasing the muscle activity. Additionally, aging affects the joint proprioception. In older subjects the joint position sense was worse than in young ones. It was also seen that knee OA patients have an even worse joint position sense than healthy elderly of a similar age. This could indicate that the age-related decline in proprioception increases the risk of OA due to reduced joint stability and increased joint stress [21].

While some age-related changes in the gait pattern like a reduced walking speed could be beneficial in reducing the risk or progression of OA [10], other changes, like muscle weakness or reduced proprioception, could increase the risk of OA, especially in the presence of additional factors such as joint laxity or pain.

Obesity has a negative impact on biomechanics, at least in weight bearing joints [22] and has been demonstrated as a risk factor for OA in large studies [23]. Interestingly, the negative effect of obesity on cartilage decay is reversible. A recent study postulated an improved cartilage quality in the medial compartment in patients with knee OA after weight loss [24]. In the elderly, obesity in combination with sarcopenia is frequently encountered, a combination which has further negative influence on OA [25]. Whether obesity causes sarcopenia in the elderly, or obesity is a consequence of reduced physical activity, remains a chicken-egg question.

4. Cartilage Decay with Age

Progressive loss of articular cartilage is considered as the hallmark of OA. Cartilage consists of chondrocytes that are embedded in a specialized extracellular matrix (ECM), which is composed of water, collagen II, and proteoglycans. Chondrocytes are quiescent nondividing cells responsible for the maintenance of cartilage homeostasis through a balanced production of catabolic and anabolic factors. During aging, senescence is induced either by progressive telomere shortening due to repeated cell division or environmental stress factors, such as oxidative damage, chronic inflammation or ultraviolet radiation [26]. Indeed, in vitro proliferation of chondrocytes from young donors differs dramatically from aged and OA donors, due to a complete lack of cell division in the latter [27]. However, as chondrocyte proliferation is scarcely observed in normal or osteoarthritic cartilage, telomere shortening is not regarded as the most likely mechanism for senescence [28]. Accumulation of oxidative damage is considered as a major player in the induction of senescence in chondrocytes. Decreased expression of oxygen radical scavengers, such as superoxide dismutase, has been demonstrated in OA cartilage [29]. There is ample evidence that age-dependent alterations in chondrocyte metabolism or signal transduction associates with progression of OA [30, 31].

These epigenetic-induced changes in chondrocytes skew the balance that dictates cartilage homeostasis and induces alterations of the ECM and cartilage integrity that promote development of OA. Loss of biomechanical function of cartilage during aging has been associated with a decrease in glycosaminoglycan content [32], which is normally deposited by chondrocytes. Besides direct modulation of the ECM by chondrocytes, age-related changes in ECM components has been proposed to mediate cartilage decay. Age-dependent accumulation and consequent cross-linking of advanced glycation end products (AGE) in collagen has been shown to increase cartilage stiffness, which makes it brittle and impairs its load-absorbing function [3]. Moreover, chondrocytes express the receptor for AGE (RAGE) and engagement of the receptor induces the production of cartilage-degrading enzymes [33]. Together, age-related changes in chondrocytes and components of ECM crucially impair the function of cartilage and its capacity to cope with mechanical or environmental stress factors, which is likely to predispose to OA development (Table 1).

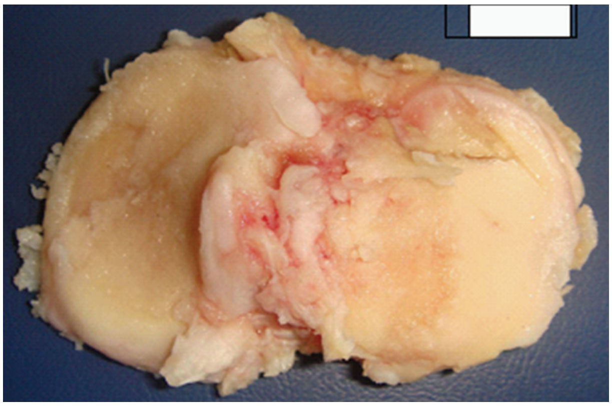
5. Subchondral Bone Adaption in Aging and the Role of Bone Mass in OA

The microarchitecture of the skeleton, notably the subchondral bone, constantly adapts to mechanical loading. Mechanotransduction processes lead to bone modeling and remodeling. Impaired joint loading patterns trigger high subchondral bone density, probably in order to avoid (micro) fractures and to ensure minimal joint function. Subchondral density can be accurately assessed by computed tomography osteoabsorptiometry (CT-OAM) [34]. Figure 2 shows a tibial plateau of a geriatric patient suffering from end stage knee OA who underwent joint replacement.

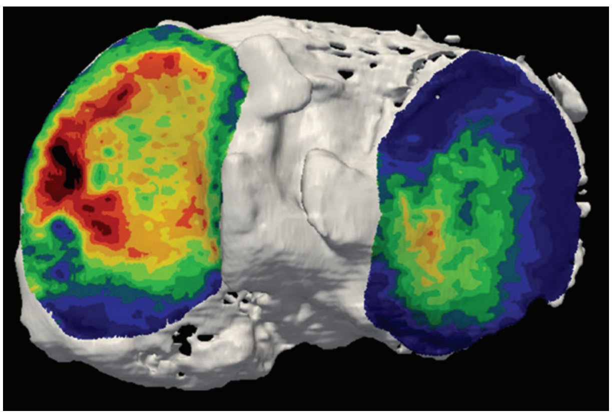
Complete cartilage loss is seen on the medial side. Below, CT-OAM was performed. Highest subchondral bone mineral density (BMD) indicated in red is observed in the region of maximal cartilage damage. On a molecular level, mechanosensors on osteoblasts have been discovered which induce interleukin-6 and -8 expression within subchondral bone affected by OA upon cyclic mechanical stimulation [35]. This

Table 1: Age-related mechanisms triggering OA.

Mechanism	Consequence
Oxidative stress	Cell senescence
Apoptosis/autophagy	Reduced regeneration capacity
Matrix protein modification (e.g., glycosylation)	Reduced elasticity, fluid content, stability
Nonenzymatic collagen cross-linking	Impaired crack-bridging potency
Sarcopenia	Reduced joint stability
Loss of proprioception	Microtrauma, Charcot-like arthropathy
Joint laxity	Microtrauma
Synovitis	Inflammatory cytokine production
Increased osteon density	Bone stiffness
Reduced circulating progenitor cells (e.g., MSC)	Impaired regeneration capacity



(a)



(b)

Figure 2: Tibial plateau of an elderly patient showing end-stage OA with complete cartilage loss (a). Computed tomography osteoabsorptiometry (CT-OAM) indicates high bone density in red (b).

is in line with the observation that increased BMD measurement of the knee correlates with clinical OA. However, the relation between generalized bone loss or osteoporosis and OA is still controversial. The MOST study has shown that high femoral neck and whole-body BMD are associated with an increased risk of OA [36]. However, osteophytes alter BMD assessment, so it remains unclear whether increased BMD is a consequence, rather than a cause of OA. Clearly, the higher bone density in patients with OA is not associated with a lower risk of vertebral or nonvertebral fractures [37].

6. Muscle Atrophy and Its Role on OA in the Elderly

Muscle atrophy can have several causes. Immobilization, malnutrition, myopathy, neurologic disorders, comorbidity of several common diseases such as cancer, congestive heart failure, COPD, or any state of chronic inflammation affect the muscle mass substantially. Also endocrinologic disorders which are generally more frequently observed in the elderly can trigger muscle atrophy, for example, low testosteron levels, diabetes mellitus, or hypothyroidism.

In contrast to secondary muscle atrophy, the commonly observed loss of muscle mass in the elderly is a syndrome referred to as "sarcopenia" [38]. Reduced muscle tissue usually starts at the age of 50 years and results in diminished muscle function. Subsequently, decreased muscle strength of the joints contribute to the pathogenesis of OA within the next decades. Fifty percent of all individuals aged over 75 suffer from sarcopenia.

The mechanistic association between OA and muscle atrophy has been clearly demonstrated. Several factors such as reduced stability, joint immobilization, restriction of range of motion but also so called arthrogenous muscle inhibition or reflex atrophy are implicated in this process. The latter is due to abnormal nociceptive afferent feedback processes, releasing neuromodulators in the spinal cord, which in turn cause a change in alpha-motoneuron excitability. In knee OA, it has been shown that quadriceps muscle strength is reduced between 20 and 40% for isometric and isokinetic contractions [39].

7. Physical Activity Preventing OA

Physiotherapy is the gold standard for OA treatment. The positive effect of physiotherapy on knee OA has been clearly demonstrated by a meta-analysis of 32 controlled trials [40]. Furthermore, physical activity seems to have preventive effects on OA. A consensus stating that performing structured strengthening exercises has a favorable effect on pain and functioning in sedentary patients with knee OA has been formulated by several organisations, for example, by the MOVE consensus [41]. In first line, physical activity in OA patients encounters clinical or subclinical muscle atrophy which has implications on joint stability [42], but also stimulates weight reduction [43].

8. The Role of Immunosenescence in OA

In the last decade, inflammation in OA has attracted increasing interest. A growing arsenal of anti-inflammatory compounds now is available which successfully works in inflammatory arthritis. On the search for disease modifying osteoarthritis drugs (DMOADs), some of those compounds have also been successfully applied to OA, at least in case reports or case series [44]. Inflammation in OA is often encountered clinically by swelling of the joint, redness and night pain. This "disease activation" in OA typically occurs in episodes, either resolving spontaneously, or sometimes by the intra-articular injection of steroids [45]. The efficacy of steroids in activated OA is well documented and safe, if applied under sterile conditions and in right intervals.

Synovial infiltration by inflammatory cells such as macrophages, mast cells or lymphocytes occurs in around 50% of the patients [6]. In MRI studies, synovitis shown with or without contrast agent correlates with pain and also radiological progression of OA. The inflammatory status of the joint is also reflected by increased pro-inflammatory cytokine levels such as TNF-alpha or IL-1 [46]. This is likely a result of the inflammatory cell infiltration in the joint and mast cell activation. Interestingly, recent findings indicate that mast cells, but also subchondral osteoblasts express mechanoreceptors which change their expression pattern upon mechanical stimulation, for example, by the expression of IL-8 [35]. But also synovial fibroblasts and even chondrocytes produce more proinflammatory cytokines and participate in inflammation.

In animal models, it has been shown that exercise leads to an increased expression of interleukin-10 in the joint [47]. IL-10 is a strong anti-inflammatory mediator and therefore likely implicated in the resolution of mechanically induced inflammation or irritation (Figure 3).

Immunosenescence is characterized by a reduced capacity of immune cells to encounter antigens and resolution of inflammation.

Recent data showed that physical activity indeed counteracts immunoscenesence [48]. Although the exact mechanism is still unclear, exercise impacts multiple aspects of the immune response including T-cell phenotype, T-cell proliferation, and as for IL-10, cytokine expression [49].

9. Cell Renewal and Stem Cell Capacity in OA

Cartilage has a low regeneration capacity due to lack of viable progenitor cells. Mesenchymal stem cells are postulated to be involved in connective tissue homeostasis and repair. The failure of repair mechanisms in connective tissues in OA is compelling. Indeed, reduced numbers of multipotent mesenchymal progenitor CD105+/CD166+ cells have been detected in the elderly. Furthermore, a lower number and lower chondrogenic and osteogenic differentiation capacity in progenitors cells from aged individuals have been demonstrated [50, 51]. Multipotent stem cells were also isolated from human cruciate ligaments suggesting their regenerative capacity also occurring in ligament structures [52]. Increased numbers of MCS are seen in the synovial fluid of patients with OA indicating a biological response [53]. These observations open the way for new therapeutic options notably in the elderly by applying those cells in the joints. Current studies investigate the possibility to obtain autologous MSC from synovial tissue in OA patients [54]. On the other hand, MSC can be obtained from other tissue, for example, adipose tissue. Notwithstanding, controlled clinical trials showing a clear efficacy of MSC treatment in OA, are still lacking.

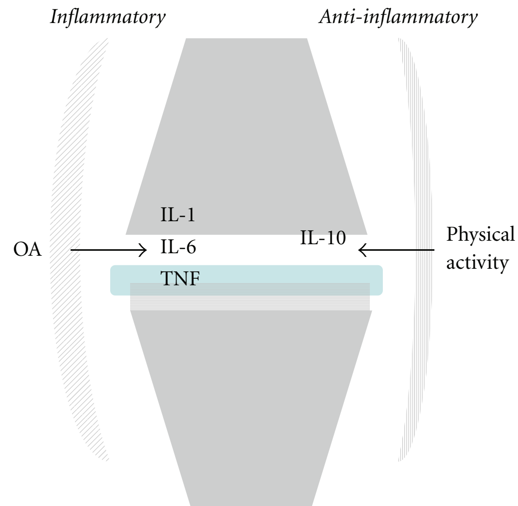


Figure 3: Cytokine expression at rest and during physical activity. OA: osteoarthritis, IL: interleukin, TNF: tumor necrosis factor.

10. Conclusion

Aging alters the human musculoskeletal system on a molecular and functional levels. Cellular renewal, matrix modification, and immunosenescence affect the regeneration capacity of connective tissue in general, but notably of bone and cartilage. This explains the important role of aging in the development and progression of OA. On the other side, several features observed during aging such as muscle atrophy, BMD, and inflammation are potentially reversible and should be counteracted, regardless of age.

References

[1] J. W. Bijlsma, F. Berenbaum, and F. P. Lafeber, "Osteoarthritis: an update with relevance for clinical practice," *The Lancet*, vol. 377, no. 9783, pp. 2115–2126, 2011.

[2] T. Aigner, J. Rose, J. Martin, and J. Buckwalter, "Aging theories of primary osteoarthritis: from epidemiology to molecular

biology," *Rejuvenation Research*, vol. 7, no. 2, pp. 134–145, 2004.

[3] N. Verzijl, J. DeGroot, Z. C. Ben et al., "Crosslinking by advanced glycation end products increases the stiffness of the collagen network in human articular cartilage: a possible mechanism through which age is a risk factor for osteoarthritis," *Arthritis & Rheumatism*, vol. 46, no. 1, pp. 114–123, 2002.

[4] J. A. Martin, S. M. Ellerbroek, and J. A. Buckwalter, "Age-related decline in chondrocyte response to insulin-like growth factor-I: the role of growth factor binding proteins," *Journal of Orthopaedic Research*, vol. 15, no. 4, pp. 491–498, 1997.

[5] E. A. Zimmermann, E. Schaible, H. Bale et al., "Age-related changes in the plasticity and toughness of human cortical bone at multiple length scales," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 108, no. 35, pp. 14416–14421, 2011.

[6] J. Sellam and F. Berenbaum, "The role of synovitis in pathophysiology and clinical symptoms of osteoarthritis," *Nature Reviews Rheumatology*, vol. 6, no. 11, pp. 625–635, 2010.

[7] M. J. Benito, D. J. Veale, O. FitzGerald, W. B. van den Berg, and B. Bresnihan, "Synovial tissue inflammation in early and late osteoarthritis," *Annals of the Rheumatic Diseases*, vol. 64, no. 9, pp. 1263–1267, 2005.

[8] C. Jin, P. Frayssinet, R. Pelker et al., "NLRP3 inflammasome plays a critical role in the pathogenesis of hydroxyapatite-associated arthropathy," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 108, no. 36, pp. 14867–14872, 2011.

[9] V. Valderrabano, V. von Tscharner, B. M. Nigg et al., "Lower leg muscle atrophy in ankle osteoarthritis," *Journal of Orthopaedic Research*, vol. 24, no. 12, pp. 2159–2169, 2006.

[10] A. Mundermann, C. O. Dyrby, and T. P. Andriacchi, "Secondary gait changes in patients with medial compartment knee osteoarthritis: increased load at the ankle, knee, and hip during walking," *Arthritis & Rheumatism*, vol. 52, no. 9, pp. 2835–2844, 2005.

[11] T. Miyazaki, M. Wada, H. Kawahara, M. Sato, H. Baba, and S. Shimada, "Dynamic load at baseline can predict radiographic disease progression in medial compartment knee osteoarthritis," *Annals of the Rheumatic Diseases*, vol. 61, no. 7, pp. 617–622, 2002.

[12] N. Foroughi, R. Smith, and B. Vanwanseele, "The association of external knee adduction moment with biomechanical variables in osteoarthritis: a systematic review," *Knee*, vol. 16, no. 5, pp. 303–309, 2009.

[13] B. G. Pietrosimone, J. Hertel, C. D. Ingersoll, J. M. Hart, and S. A. Saliba, "Voluntary quadriceps activation deficits in patients with tibiofemoral osteoarthritis: a meta-analysis," *PM & R*, vol. 3, no. 2, pp. 153–162, 2011.

[14] E. M. Roos, W. Herzog, J. A. Block, and K. L. Bennell, "Muscle weakness, afferent sensory dysfunction and exercise in knee osteoarthritis," *Nature Reviews Rheumatology*, vol. 7, no. 1, pp. 57–63, 2011.

[15] N. A. Segal, J. C. Torner, D. Felson et al., "Effect of thigh strength on incident radiographic and symptomatic knee osteoarthritis in a longitudinal cohort," *Arthritis Care and Research*, vol. 61, no. 9, pp. 1210–1217, 2009.

[16] M. V. Hurley, "The role of muscle weakness in the pathogenesis of osteoarthritis," *Rheumatic Disease Clinics of North America*, vol. 25, no. 2, pp. 283–298, 1999.

[17] R. W. Bohannon and A. A. Williams, "Normal walking speed: a descriptive meta-analysis," *Physiotherapy*, vol. 97, no. 3, pp. 182–189, 2011.

[18] J. O. Judge, R. B. Davis III, and S. Ounpuu, "Step length reductions in advanced age: the role of ankle and hip kinetics," *Journals of Gerontology A*, vol. 51, no. 6, pp. M303–M312, 1996.

[19] H. B. Menz, S. R. Lord, and R. C. Fitzpatrick, "Age-related differences in walking stability," *Age and Ageing*, vol. 32, no. 2, pp. 137–142, 2003.

[20] K. S. Rudolph, L. C. Schmitt, and M. D. Lewek, "Age-related changes in strength, joint laxity, and walking patterns: are they related to knee osteoarthritis?" *Physical Therapy*, vol. 87, no. 11, pp. 1422–1432, 2007.

[21] Y. C. Pai, W. Z. Rymer, R. W. Chang, and L. Sharma, "Effect of age and osteoarthritis on knee proprioception," *Arthritis & Rheumatism*, vol. 40, no. 12, pp. 2260–2265, 1997.

[22] J. Runhaar, B. W. Koes, S. Clockaerts, and S. M. Bierma-Zeinstra, "A systematic review on changed biomechanics of lower extremities in obese individuals: a possible role in development of osteoarthritis," *Obesity Reviews*, vol. 12, no. 12, pp. 1071–1082, 2011.

[23] D. T. Felson, J. J. Anderson, A. Naimark, A. M. Walker, and R. F. Meenan, "Obesity and knee osteoarthritis. The Framingham study," *Annals of Internal Medicine*, vol. 109, no. 1, pp. 18–24, 1988.

[24] A. Anandacoomarasamy, S. Leibman, G. Smith et al., "Weight loss in obese people has structure-modifying effects on medial but not on lateral knee articular cartilage," *Annals of the Rheumatic Diseases*, vol. 71, no. 1, pp. 26–32, 2012.

[25] D. L. Waters and R. N. Baumgartner, "Sarcopenia and obesity," *Clinics in Geriatric Medicine*, vol. 27, no. 3, pp. 401–421, 2011.

[26] J. Campisi and F. D'Adda di Fagagna, "Cellular senescence: when bad things happen to good cells," *Nature Reviews Molecular Cell Biology*, vol. 8, no. 9, pp. 729–740, 2007.

[27] B. Dozin, M. Malpeli, L. Camardella, R. Cancedda, and A. Pietrangelo, "Response of young, aged and osteoarthritic human articular chondrocytes to inflammatory cytokines: molecular and cellular aspects," *Matrix Biology*, vol. 21, no. 5, pp. 449–459, 2002.

[28] T. Aigner, M. Hemmel, D. Neureiter et al., "Apoptotic cell death is not a widespread phenomenon in normal aging and osteoarthritis human articular knee cartilage: a study of proliferation, programmed cell death (apoptosis), and viability of chondrocytes in normal and osteoarthritic human knee cartilage," *Arthritis & Rheumatism*, vol. 44, no. 6, pp. 1304–1312, 2001.

[29] E. Regan, J. Flannelly, R. Bowler et al., "Extracellular superoxide dismutase and oxidant damage in osteoarthritis," *Arthritis & Rheumatism*, vol. 52, no. 11, pp. 3479–3491, 2005.

[30] E. N. B. Davidson, D. F. Remst, E. L. Vitters et al., "Increase in ALK1/ALK5 ratio as a cause for elevated MMP-13 expression in osteoarthritis in humans and mice," *Journal of Immunology*, vol. 182, no. 12, pp. 7937–7945, 2009.

[31] J. D. Cravero, C. S. Carlson, H. J. Im, R. R. Yammani, D. Long, and R. F. Loeser, "Increased expression of the Akt/PKB inhibitor TRB3 in osteoarthritic chondrocytes inhibits insulin-like growth factor 1-mediated cell survival and proteoglycan synthesis," *Arthritis & Rheumatism*, vol. 60, no. 2, pp. 492–500, 2009.

[32] M. M. Temple, W. C. Bae, M. Q. Chen et al., "Age- and site-associated biomechanical weakening of human articular cartilage of the femoral condyle," *Osteoarthritis and Cartilage*, vol. 15, no. 9, pp. 1042–1052, 2007.

[33] S. S. Nah, I. Y. Choi, B. Yoo, Y. G. Kim, H. B. Moon, and C. K. Lee, "Advanced glycation end products increases matrix metalloproteinase-1, -3, and -13, and TNF-α in human

osteoarthritic chondrocytes," *The FEBS Letters*, vol. 581, no. 9, pp. 1928–1932, 2007.

[34] M. Muller-Gerbl, R. Putz, N. Hodapp, E. Schulte, and B. Wimmer, "Computed tomography-osteoabsorptiometry for assessing the density distribution of subchondral bone as a measure of long-term mechanical adaptation in individual joints," *Skeletal Radiology*, vol. 18, no. 7, pp. 507–512, 1989.

[35] C. Sanchez, M. A. Deberg, A. Bellahcene et al., "Phenotypic characterization of osteoblasts from the sclerotic zones of osteoarthritic subchondral bone," *Arthritis & Rheumatism*, vol. 58, no. 2, pp. 442–455, 2008.

[36] M. C. Nevitt, Y. Zhang, M. K. Javaid et al., "High systemic bone mineral density increases the risk of incident knee OA and joint space narrowing, but not radiographic progression of existing knee OA: the MOST study," *Annals of the Rheumatic Diseases*, vol. 69, no. 1, pp. 163–168, 2010.

[37] N. K. Arden, S. Crozier, H. Smith et al., "Knee pain, knee osteoarthritis, and the risk of fracture," *Arthritis & Rheumatism*, vol. 55, no. 4, pp. 610–615, 2006.

[38] I. H. Rosenberg, "Sarcopenia: origins and clinical relevance," *Clinics in Geriatric Medicine*, vol. 27, no. 3, pp. 337–339, 2011.

[39] B. S. Hassan, S. A. Doherty, S. Mockett, and M. Doherty, "Effect of pain reduction on postural sway, proprioception, and quadriceps strength in subjects with knee osteoarthritis," *Annals of the Rheumatic Diseases*, vol. 61, no. 5, pp. 422–428, 2002.

[40] M. Fransen and S. McConnell, "Exercise for osteoarthritis of the knee," *Cochrane Database of Systematic Reviews*, no. 4, Article ID CD004376, 2008.

[41] E. Roddy, W. Zhang, M. Doherty et al., "Evidence-based recommendations for the role of exercise in the management of osteoarthritis of the hip or knee—the MOVE concensus," *Rheumatology*, vol. 44, no. 1, pp. 67–73, 2005.

[42] C. Slemenda, D. K. Heilman, K. D. Brandt et al., "Reduced quadriceps strength relative to body weight: a risk factor for knee osteoarthritis in women?" *Arthritis & Rheumatism*, vol. 41, no. 11, pp. 1951–1959, 1998.

[43] M. A. Davis, J. M. Neuhaus, W. H. Ettinger, and W. H. Mueller, "Body fat distribution and osteoarthritis," *American Journal of Epidemiology*, vol. 132, no. 4, pp. 701–707, 1990.

[44] S. B. Abramson and Y. Yazici, "Biologics in development for rheumatoid arthritis: relevance to osteoarthritis," *Advanced Drug Delivery Reviews*, vol. 58, no. 2, pp. 212–225, 2006.

[45] C. T. Hepper, J. J. Halvorson, S. T. Duncan, A. J. Gregory, W. R. Dunn, and K. P. Spindler, "The efficacy and duration of intra-articular corticosteroid injection for knee osteoarthritis: a systematic review of level I studies," *Journal of the American Academy of Orthopaedic Surgeons*, vol. 17, no. 10, pp. 638–646, 2009.

[46] M. Kapoor, J. Martel-Pelletier, D. Lajeunesse, J. P. Pelletier, and H. Fahmi, "Role of proinflammatory cytokines in the pathophysiology of osteoarthritis," *Nature Reviews Rheumatology*, vol. 7, no. 1, pp. 33–42, 2011.

[47] I. C. Helmark, U. R. Mikkelsen, J. Borglum et al., "Exercise increases interleukin-10 levels both intraarticularly and perisynovially in patients with knee osteoarthritis: a randomized controlled trial," *Arthritis Research & Therapy*, vol. 12, no. 4, article R126, 2010.

[48] D. S. Senchina and M. L. Kohut, "Immunological outcomes of exercise in older adults," *Clinical Interventions in Aging*, vol. 2, no. 1, pp. 3–16, 2007.

[49] D. C. Nieman, D. A. Henson, G. Gusewitch et al., "Physical activity and immune function in elderly women," *Medicine and Science in Sports and Exercise*, vol. 25, no. 7, pp. 823–831, 1993.

[50] H. X. Chang, L. Yang, Z. Li, G. Chen, and G. Dai, "Age-related biological characterization of mesenchymal progenitor cells in human articular cartilage," *Orthopedics*, vol. 34, no. 8, pp. e382–e388, 2011.

[51] C. de Bari, T. B. Kurth, and A. Augello, "Mesenchymal stem cells from development to postnatal joint homeostasis, aging, and disease," *Birth Defects Research C*, vol. 90, no. 4, pp. 257–271, 2010.

[52] M. T. Cheng, H. W. Yang, T. H. Chen, and O. K. Lee, "Isolation and characterization of multipotent stem cells from human cruciate ligaments," *Cell Proliferation*, vol. 42, no. 4, pp. 448–460, 2009.

[53] I. Sekiya, M. Ojima, S. Suzuki et al., "Human mesenchymal stem cells in synovial fluid increase in the knee with degenerated cartilage and osteoarthritis," *Journal of Orthopaedic Research*, vol. 30, no. 6, pp. 943–949, 2012.

[54] M. J. Kim, M. J. Son, M. Y. Son et al., "Generation of human induced pluripotent stem cells from osteoarthritis patient-derived synovial cells," *Arthritis & Rheumatism*, vol. 63, no. 10, pp. 3010–3021, 2011.

Hindawi Publishing Corporation
Journal of Aging Research
Volume 2012, Article ID 345237, 8 pages
doi:10.1155/2012/345237

Review Article

Total Ankle Replacement for Treatment of End-Stage Osteoarthritis in Elderly Patients

Beat Hintermann, Markus Knupp, Lukas Zwicky, and Alexej Barg

Clinic of Orthopaedic Surgery, Kantonsspital Liestal, 4410 Liestal, Switzerland

Correspondence should be addressed to Alexej Barg, alexejbarg@mail.ru

Received 28 February 2012; Accepted 19 April 2012

Academic Editor: Iris Reuter

End-stage osteoarthritis of the ankle is a disabling problem, particularly in elderly patients who experience an overall loss of mobility and functional impairment and who then need compensatory adaption. Ankle arthrodesis, which has been demonstrated to provide postoperative pain relief and hindfoot stability, leaves the patient with a stiff foot and gait changes. For elderly patient, these changes may be more critical than generally believed. Additionally, the long duration of healing and rehabilitation process needed for ankle arthrodesis may be problematic in the elderly. In contrast to ankle arthrodesis, total ankle replacement has significant advantages including a less strenuous postoperative rehabilitation and preservation of ankle motion which supports physiological gait. Recently, total ankle replacement has evolved as a safe surgical treatment in patients with end-stage ankle osteoarthritis with reliable mid- to long-term results. Total ankle replacement needs less immobilization than arthrodesis and does allow for early weight-bearing and should be considered as a treatment option of first choice in many elderly patients with end-stage osteoarthritis of the ankle, especially in elderly patients with lower expectations and physical demands.

1. Introduction

The ankle joint has a much lower incidence of symptomatic osteoarthritis (OA) compared to other major joints of the lower extremity [1]. This, despite the facts that the articular cartilage in the ankle experiences the greatest contact force per unit area of any major joint in the body and the ankle joint is one of the most commonly injured areas in orthopaedic surgery [1–3]. However, degenerative OA of the ankle is a constantly growing problem: currently approximately 1% of the world's adult population is affected by ankle OA leading to significant mental and physical disabilities [4].

Trauma is the primary cause of ankle OA [5, 6]. Valderrabano et al. reviewed 406 ankles that presented with symptomatic end-stage ankle arthritis and found 78% secondary to previous trauma [6]. Patients usually presented with a lower leg fracture in the history, but also repetitive ligamentous injuries of the hindfoot complex may lead to degenerative OA of the ankle [7]. Primary arthritis (possibly secondary to misalignment) occurred in up to 10% of all patients. The remaining 10–15% of patients developed secondary ankle OA due to the following underlying diseases: rheumatoid disease, hemochromatosis [8], haemophilia [9], gout [10], clubfoot [11], aseptic talar necrosis, and after joint infection.

Ankle arthrodesis remains an important treatment option in patients with end-stage ankle OA [12, 13]. After a successful fusion, patients consistently report both, pain relief and improved mobility [12]. However, many clinical studies describe short- and long-term problems following ankle arthrodesis including acute or chronic infection, delayed union, and decreased functional ability [12, 14]. For the majority of patients who achieve full healing of the arthrodesis, the time of convalescence can be difficult: time duration to achieve complete bone healing may range from 12 to 20 weeks. The postoperative recovery involves some form of immobilization and restricted weight-bearing activities, which can cause significant leg muscle atrophy. Even after the ankle arthrodesis is fully healed, some patients may develop profound dysfunction in the long term. Many authors make note of significant limitations with walking inclines, accommodating uneven ground, driving, and athletic activities [14–16]. Formal gait studies after ankle arthrodesis show decreased cadence and stride with decreased motion of the midfoot and hindfoot complexes [17–19]. Gait and function may be also significantly affected if patients develop adjacent joint OA. Coester et al. found in their long-term clinical observational study that majority of patients who underwent an ankle arthrodesis has developed degenerative changes in the ipsilateral foot but not the knee [20]. Similar findings were observed in the long-term study by Fuchs et al., showing deficits in the functional outcome, limitation in the activities of daily living, and radiological changes in the adjacent joints in patients 20 years after ankle arthrodesis [16].

Although ankle arthrodesis is a valid treatment option for end-stage ankle arthritis, its risks and sequelae cannot be ignored. Total ankle replacement (TAR) using current prosthesis designs have evolved to reliable treatment option in patients with end-stage ankle OA. Therefore, ankle fusion is no longer the "gold standard" therapy in this patient cohort [21]. Despite significant progress, concerns still persist related to the feasibility of TAR in patients with bad bone and soft tissue quality, as is often the case in elderly after previous trauma or systemic disease.

The purpose of this paper is therefore to evaluate the potential benefits of TAR in elderly patients with age over 60 years [22], in particular to its advantages with regard to ankle arthrodesis.

2. Biomechanics and Gait Analysis

The biomechanics of gait in healthy patients with nonarthritic ankles are clearly different when compared with patients with arthritic, fused, and replaced ankles [19, 21, 23–25].

In choosing between fusion and TAR, benefits in favour of TAR include restoring ankle motion, improving gait biomechanics, and avoiding advanced adjacent joint degeneration more commonly seen following ankle fusion [19, 21, 23–28]. Restoring or at least improving upon gait biomechanics of patients with end-stage ankle arthritis is one of the main goals of surgical treatment for this disease.

Ankle fusion and TAR patients can be expected to have slower gait velocities when compared with healthy control groups but faster speeds when compared with their preoperative arthritic ankle condition [17, 19, 23, 25]. TAR patients exhibit a fairly symmetric gait, while ankle fusion patients require significant compensatory mechanisms to obtain a steady, symmetric gait, including increased midfoot joint motion as well as increased range of motion of the ipsilateral knee [17, 19, 23, 25].

In summary, though patients with a fused ankle can be expected to have a reasonably efficient gait, TAR may offer the patient a more normal gait with less negative impact on segmental motion of the whole lower limb and stress concentration on adjacent joints.

3. Surgical Technique and Postoperative Care

Meticulous preoperative planning is the main step for success of TAR [29]. Evaluation in the outpatient clinic entails a detailed history taking, including an evaluation of previous infection, trauma, surgeries, failure or success of treatments, location of pain, social circumstances, previous and current activity level, expectations of treatment, tolerance for revision surgery and general health, especially as it relates to a history of neuropathy and/or diabetes. Also, all previous medical reports (e.g., surgery reports) and imaging studies should be completely collected.

The routine physical examination includes careful inspection of the entire foot and ankle. Hindfoot stability should be assessed manually with the patient sitting. Ankle alignment and range of motion are assessed with the patient standing. Range of motion is determined clinically with a goniometer placed along the lateral border of the leg and foot [30, 31]. Assessment of the subtalar motion and palpation of sinus tarsi may help to exclude subtalar OA. The patients gait is observed clinically and then analyzed using pedobarography in most patients [32]. All affected ankles need to be preoperatively evaluated based on weight-bearing radiographs in three planes. The Saltzman view is used for standardized assessment of varus and valgus deformity of the hindfoot [33]. Single-photon emission-computed tomography (SPECT-CT) can be performed for an accurate assessment and localization of degenerative changes in the adjacent joints [34, 35].

Most manufactures of ankle prostheses provide reliable instrumentation to perform the appropriate bone cuts and to prepare the resection surfaces to accommodate the prosthesis components. Most surgeons use an anterior approach for exposure of the ankle (Figure 1). Careful dissection of soft tissues and avoidance of any unnecessary soft tissue retraction are keys to success to avoid postoperative wound healing complications. Release of any soft tissue contracture is mandatory to gain joint motion, but also to balance the talus properly within the ankle mortise. Heel cord lengthening may be advised in some cases of equines contracture; its use should be very restrictive as patients will often complain about longstanding soft tissue pain along the tendon and loss of plantar flexion power.

Combined peritalar and ankle arthritis, and complex misalignment of the ankle joint complex are complex and challenging clinical entities [36–40]. Combined peritalar and ankle arthritis and varus/valgus preoperative deformity can be successfully treated with TAR in selected cases but need in most instances additional procedures at the same time [39]. Attention to detail, a meticulous preoperative evaluation, and a carefully planned or staged surgery optimize the chances of a successful result [39, 41].

After surgery, the foot is protected by a splint. When the wound condition is proper, typically 2 to 4 days after surgery, the foot is placed in a short leg weight-bearing cast or a walker for 6 weeks, and a brace may be used for 4 to 6 additional weeks. Most importantly, the patient is allowed for full weight-bearing from the beginning, with only exception where additional surgeries do not allow it (e.g., correcting tibial osteotomies). After the cast is removed, the rehabilitation program was started, with gradual return to full activities as tolerated. Radiological controls are made 6 weeks,

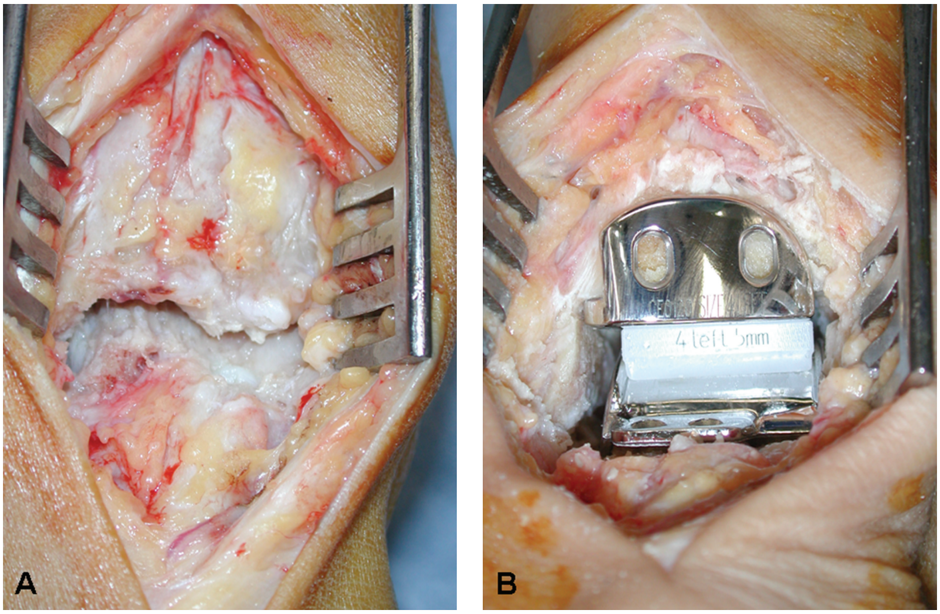


Figure 1: Intraoperative pictures of (A) exposure of arthritic ankle, and (B) after implantation of prosthesis. 66-year-old female patient after septic arthritis.

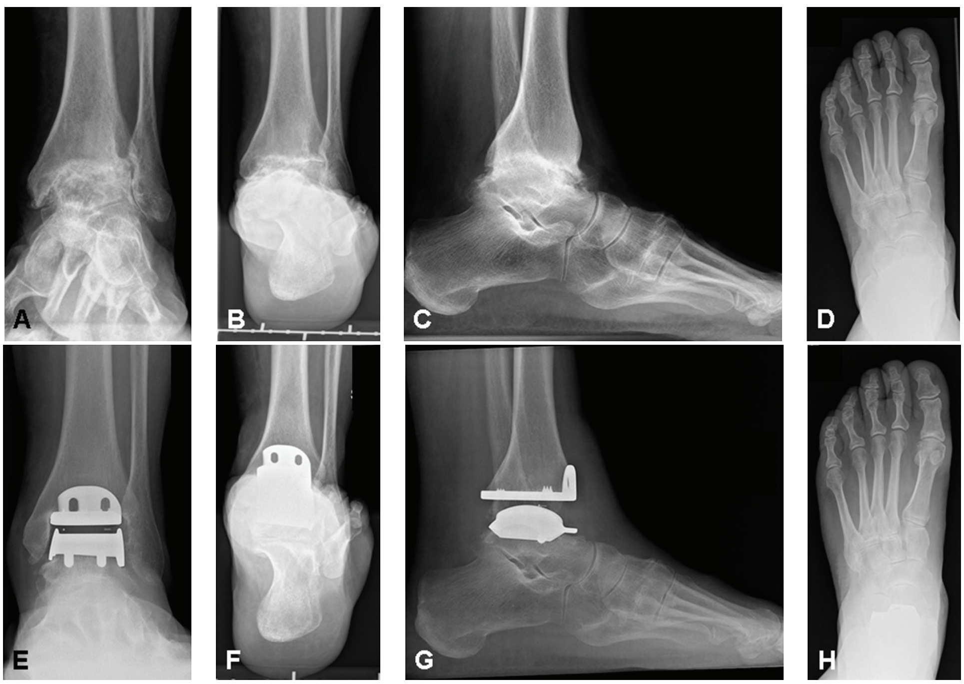


Figure 2: Radiologic evaluation preoperatively (A–D) and after 5 years (E–H): AP view of the ankle (A, E), Saltzman alignment view (B, F), lateral view (C, G), and AP view of the foot (D, H). Same patient as in Figure 1.

4 months, and 12 months after surgery and then annually (Figure 2).

4. Results

In the orthopaedic literature, there are very few studies that compare implants head to head that are either Level I or Level II, and the superiority of an implant design over another cannot be supported by any available data from Level IV studies [42]. The experiences of several national joint registries have been published previously [43–46]. No statistically significant risk factors (e.g., age, gender, type of prosthesis, underlying etiology) have been identified as influencing survivorship of prosthesis components in

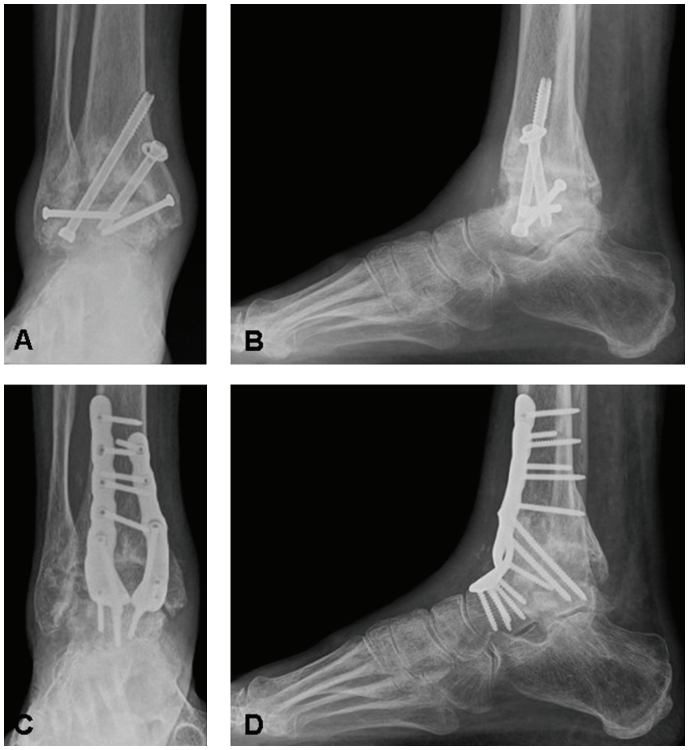


Figure 3: 71-year-old male patient, 5 years after attempted fusion of the ankle (A-B). He was never pain-free. His orthopaedic surgeon led him in believe that the ankle was completely fused. One year after revision arthrodesis using a rigid anterior plate fixation, the fusion was healed and the patient was pain-free (C-D).

Norwegian Arthroplasty Register [43], Finnish Arthroplasty Register [46], and New Zealand National Joint Registry [45]. In the Swedish Ankle Arthroplasty Register, lower age at TAR surgery was associated with increased risk of revision whereas preoperative diagnosis or gender did not [44]. Prosthesis misalignment and aseptic loosening have been consistently found to be the most common cause for prosthesis revision [43–46]. A recent systematic review of the literature including 13 Level IV studies with 1105 TARs showed the overall failure rate of approximately 10% at 5 years with a wide range between 0% and 32% [42].

Based on our own registry on 394 ankles (female, 199; male 195; mean age 59.7 [25.3–90.0] years) with a minimal followup of 5 years, our revision free rate at 5 years was 95.1% and 85.6% at 10 years. The revision rate was higher in posttraumatic osteoarthritis than in primary or systemic osteoarthritis. There was no difference in outcome between female and male patients. Over 60-year-old patients evidenced, fewer complications than those patients of less than 60 years old, and they had also fewer revisions.

5. Complications

Numerous reports describing several techniques for ankle arthrodesis report the fusion rate of 85% or greater, which may depend on the presence of infection, deformity, avascular necrosis, and nonunion [12, 47]. However, only in few studies, a CT scan has been used to assess the postoperative osseous healin; therefore, the reported fusion rate of up to 100% may be overestimated. Since 2008, a total of 38 patients presented in author's outpatient clinic with persistent pain after ankle arthrodesis related to a nonunion (Figure 3), 31 patients (81.6%) were thought to have undergone successful ankle fusion by their treating orthopaedic surgeon.

The incidence of nonunion in ankle arthrodesis to a certain extent depends on the surgical technique used [12, 47]. Open procedures involve greater soft-tissue stripping than limited open or arthroscopic techniques. Poor bone quality as typically is the case in elderly patients remains a challenge for achieving primary stability for both external and internal fixation. Newer techniques with rigid plate fixation have shown superior results but may be associated with a higher risk of soft tissue complications or need for hardware removal due to discomfort [13, 48, 49].

Beside nonunion, mal-union of the fused ankle is another one of the most disabling conditions. The most common mal-union is due to unphysiological plantar flexed position [31]. In addition to consecutively developed metatarsalgia, the longstanding plantar flexed mal-union may be a risk factor for development of degenerative changes in subtalar and/or talonavicular joints. Fusion in dorsiflexion, by contrast, may lead to "back-kneeing" or genu recurvatum. This, in turn, places the patient's center of gravity in front of the weight-bearing axis causing vaulting over the improperly

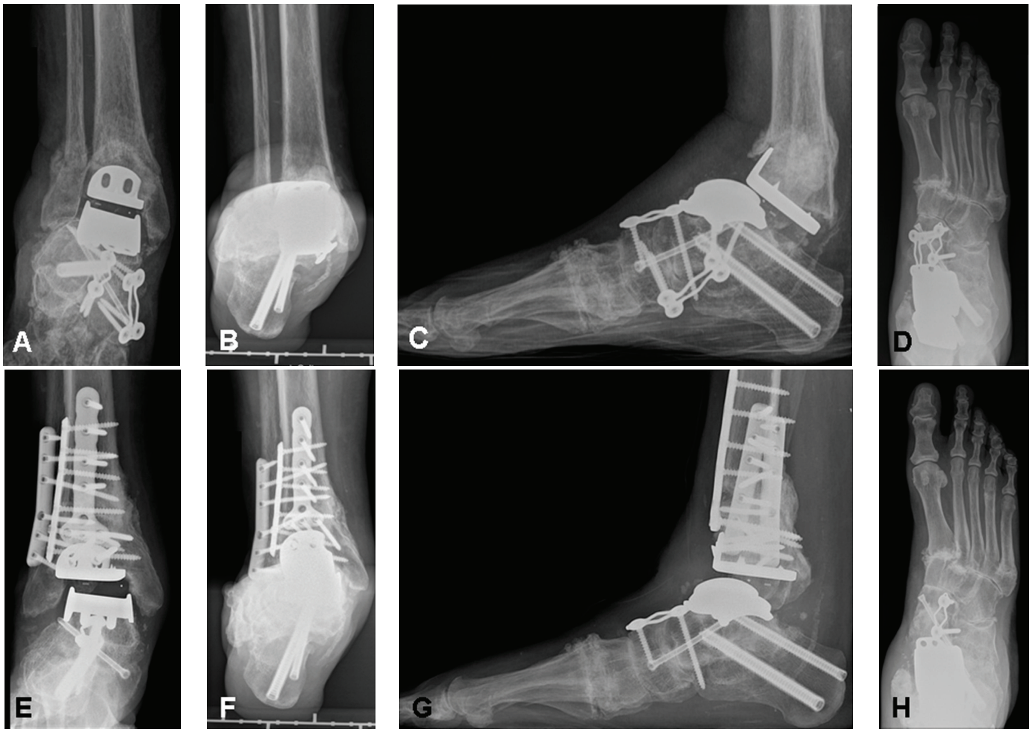


Figure 4: Periprosthetic fracture in a 61-year-old female patient with rheumatoid arthritis after struggling on the stairs. Marked angulation into valgus (A-B) and recurvatum (C), with supination of the foot (D). Uneventful evolution after internal fixation. One year afterwards, the ankle was stable and the patient was able to walk without any pain, though the ankle was still in slight valgus (E-F). Correct situation in lateral view (G) and AP view (H) of the foot.

positioned foot. Varus or valgus malunion may also present problems but usually only if severe.

In preserving joint motion, TAR offers an excellent alternative to arthrodesis and its sequelae [50–52]. The early complications after TAR include break down of wounds and superficial and deep infection [53, 54]. With current techniques and implants, the risk of primary loosening has dramatically decreased. Nevertheless, in the presence of poor bone quality, a successfully replaced ankle may be susceptible to periprothetic fracture during early remodelling phase (Figure 4).

The main risk of failure after total ankle replacement results from not achieving a balanced ankle joint complex [55]. As a majority part of end-stage osteoarthritic ankles will present with associated problems such as misaligned hindfoot, varus or valgus tilt of talus within the mortise, instability, or soft tissue contractures, the surgeon must be familiar with addressing these associated problems to get a successful replaced ankle [39, 56]. Surgeon's experience may thus play a superior role for success in TAR [57–60].

The use of TAR in elderly patients still remains controversial in orthopaedic surgery [52]. Kofoed and Lundberg-Jensen [61] have performed a prospective study reporting 100 consecutive cases of patients with osteoarthritis or rheumatoid arthritis with a followup up to 15 years. In all patients, Scandinavian Total Ankle Replacement has been used. All patients were divided into two groups: younger and older than 50 years. The authors found that TAR is a safe and reliable procedure for both, younger and elderly patients with 75.0% and 80.6% survivorship at 6 years, respectively [61]. Several other studies have shown a more favourable outcome of TAR in patients with rheumatoid arthritis and elderly low-demand patients with degeneration ankle arthritis [62–67]. Spirt et al. [68] have analyzed the cause and frequency of reoperation and failure after 306 primary total ankle arthroplasties using DePuy Agility prosthesis. Age at the time of the primary TAR was the only covariate that had an impact on the hazard of reoperation and failure: each one-year increase in age corresponded with a 1.9% relative decrease in the hazard of reoperation and 3.5% decrease in the hazard of failure [68].

The ideal patient for TAR continues to be debated within the orthopaedic foot and ankle surgeons [52]. However, in the most studies, the ideal candidate for TAR has been identified as reasonably mobile, *middle aged or older patient*, with no obesity or overweight and well aligned and stable hindfoot [52, 62, 69–76].

6. Conclusions

TAR has evolved as a reliable and safe alternative to arthrodesis in the treatment of end-stage ankle osteoarthritis [50, 53, 77]. Reduction in device constraint realized by the contemporary prosthetic designs in comparison with the first generation devices and improved instrumentation has markedly contributed to this higher success. Clinical longevity of TAR is dependent upon a correct balance between the intrinsic mobility allowed by the design and the presenting pathology of the patient [55]. This is further influenced by the ability of the surgeon to appropriately balance the soft tissue constraints and correctly align the components [57–60]. Despite improvement in designing appropriate surgical training, experience and technique will ultimately determine the results of total ankle arthroplasty [57–60].

The elderly patient may benefit more by TAR then the alternative ankle arthrodesis. First, postoperative rehabilitation after TAR is easier than that after ankle arthrodesis, allowing for full weight-bearing from the beginning. Immobilization and protection time is usually also markedly shorter for TAR. Thus, loss of articular and muscular function may be less than that after ankle arthrodesis [78–80]. Second, TAR may better restore hindfoot biomechanics, resulting in less gait adoptions and functional impairment [81]. Finally, TAR is in particular promising for elderly patients as the physical demands are, in general, lower. In summary, TAR has yielded to a valuable alternative to ankle arthrodesis and thus can be recommended in elderly patients as a very promising option to regain life quality and function.

References

[1] D. D. Anderson, S. Chubinskaya, F. Guilak et al., "Posttraumatic osteoarthritis: improved understanding and opportunities for early intervention," *Journal of Orthopaedic Research*, vol. 29, no. 6, pp. 802–809, 2011.

[2] M. Knupp, V. Valderrabano, and B. Hintermann, "Anatomical and biomechanical aspects of total ankle replacement," *Orthopade*, vol. 35, no. 5, pp. 489–494, 2006.

[3] M. Müller-Gerbl, "Anatomy and biomechanics of the ankle joint," *Orthopade*, vol. 30, no. 1, pp. 3–11, 2001.

[4] M. Glazebrook, T. Daniels, A. Younger et al., "Comparison of health-related quality of life between patients with end-stage ankle and hip arthrosis," *Journal of Bone and Joint Surgery Series A*, vol. 90, no. 3, pp. 499–505, 2008.

[5] C. L. Saltzman, M. L. Salamon, G. M. Blanchard et al., "Epidemiology of ankle arthritis: report of a consecutive series of 639 patients from a tertiary orthopaedic center," *The Iowa Orthopaedic Journal*, vol. 25, pp. 44–46, 2005.

[6] V. Valderrabano, M. Horisberger, I. Russell, H. Dougall, and B. Hintermann, "Etiology of ankle osteoarthritis," *Clinical Orthopaedics and Related Research*, vol. 467, no. 7, pp. 1800–1806, 2009.

[7] V. Valderrabano, B. Hintermann, M. Horisberger, and S. F. Tak, "Ligamentous posttraumatic ankle osteoarthritis," *American Journal of Sports Medicine*, vol. 34, no. 4, pp. 612–620, 2006.

[8] A. Barg, A. Elsner, D. Hefti, and B. Hintermann, "Total ankle arthroplasty in patients with hereditary hemochromatosis," *Clinical Orthopaedics and Related Research*, vol. 469, no. 5, pp. 1427–1435, 2011.

[9] A. Barg, A. Elsner, D. Hefti, and B. Hintermann, "Haemophilic arthropathy of the ankle treated by total ankle replacement: a case series," *Haemophilia*, vol. 16, no. 4, pp. 647–655, 2010.

[10] A. Barg, M. Knupp, A. L. Kapron, and B. Hintermann, "Total ankle replacement in patients with gouty arthritis," *Journal of Bone and Joint Surgery*, vol. 93, no. 4, pp. 357–366, 2011.

[11] M. Knupp, A. Barg, L. Bolliger, and B. Hintermann, "Supramalleolar osteotomies for the treatment of overcorrected clubfoot deformity," *Journal of Bone and Joint Surgery*. In press.

[12] A. Nihal, R. E. Gellman, J. M. Embil, and E. Trepman, "Ankle arthrodesis," *Foot and Ankle Surgery*, vol. 14, no. 1, pp. 1–10, 2008.

[13] C. Plaass, M. Knupp, A. Barg, and B. Hintermann, "Anterior double plating for rigid fixation of isolated tibiotalar arthrodesis," *Foot and Ankle International*, vol. 30, no. 7, pp. 631–639, 2009.

[14] J. M. Mazur, E. Schwartz, and S. R. Simon, "Ankle arthrodesis. Long-term follow-up with gait analysis," *Journal of Bone and Joint Surgery Series A*, vol. 61, no. 7, pp. 964–975, 1979.

[15] G. N. Boobbyer, "The long-term results of ankle arthrodesis," *Acta Orthopaedica Scandinavica*, vol. 52, no. 1, pp. 107–110, 1981.

[16] S. Fuchs, C. Sandmann, A. Skwara, and C. Chylarecki, "Quality of life 20 years arthrodesis of the ankle," *Journal of Bone and Joint Surgery Series B*, vol. 85, no. 7, pp. 994–998, 2003.

[17] P. Piriou, P. Culpan, M. Mullins, J. N. Cardon, D. Pozzi, and T. Judet, "Ankle replacement versus arthrodesis: a comparative gait analysis study," *Foot and Ankle International*, vol. 29, no. 1, pp. 3–9, 2008.

[18] K. M. Schweitzer and S. G. Parekh, "Comparison of gait biomechanics: ankle fusion versus ankle replacement," *Seminars in Arthroplasty*, vol. 21, no. 4, pp. 223–229, 2010.

[19] W. L. Wu, F. C. Su, Y. M. Cheng, P. J. Huang, Y. L. Chou, and C. K. Chou, "Gait analysis after ankle arthrodesis," *Gait and Posture*, vol. 11, no. 1, pp. 54–61, 2000.

[20] L. M. Coester, C. L. Saltzman, J. Leupold, and W. Pontarelli, "Long-term results following ankle arthrodesis for post-traumatic arthritis," *Journal of Bone and Joint Surgery Series A*, vol. 83, no. 2, pp. 219–228, 2001.

[21] C. L. Saltzman, R. A. Mann, J. E. Ahrens et al., "Prospective controlled trial of STAR total ankle replacement versus ankle fusion: initial results," *Foot and Ankle International*, vol. 30, no. 7, pp. 579–596, 2009.

[22] World Health Organization, "Definition of an older or elderly person," 2012, http://www.who.int/healthinfo/survey/ageingdefnolder/en/index.html.

[23] J. W. Brodsky, F. E. Polo, S. C. Coleman, and N. Bruck, "Changes in gait following the scandinavian total ankle replacement," *Journal of Bone and Joint Surgery Series A*, vol. 93, no. 20, pp. 1890–1896, 2011.

[24] C. Nüesch, V. Valderrabano, C. Huber, T. V. von, and G. Pagenstert, "Gait patterns of asymmetric ankle osteoarthritis patients," *Clinical Biomechanics*. In press.

[25] V. Valderrabano, B. M. Nigg, V. von Tscharner, D. J. Stefanyshyn, B. Goepfert, and B. Hintermann, "Gait analysis in ankle osteoarthritis and total ankle replacement," *Clinical Biomechanics*, vol. 22, no. 8, pp. 894–904, 2007.

[26] V. Valderrabano, B. Hintermann, B. M. Nigg, D. Stefanyshyn, and P. Stergiou, "Kinematic changes after fusion and total replacement of the ankle—part 2: movement transfer," *Foot and Ankle International*, vol. 24, no. 12, pp. 888–896, 2003.

[27] V. Valderrabano, B. Hintermann, B. M. Nigg, D. Stefanyshyn, and P. Stergiou, "Kinematic changes after fusion and total replacement of the ankle—part 1: range of motion," *Foot and Ankle International*, vol. 24, no. 12, pp. 881–887, 2003.

[28] V. Valderrabano, B. Hintermann, B. M. Nigg, D. Stefanyshyn, and P. Stergiou, "Kinematic changes after fusion and total replacement of the ankle—part 3: talar movement," *Foot and Ankle International*, vol. 24, no. 12, pp. 897–900, 2003.

[29] B. Hintermann and A. Barg, "The HINTEGRA total ankle arthroplasty," in *Operative Techniques in Orthopaedic Surgery*, S. W. Wiesel, Ed., pp. 4022–4031, Lippincott Williams & Wilkins, 2010.

[30] A. Barg, H. B. Henninger, M. Knupp, and B. Hintermann, "Simultaneous bilateral total ankle replacement using a 3-component prosthesis: outcome in 26 patients followed for 2–10 years," *Acta Orthopaedica*, vol. 82, no. 6, pp. 704–710, 2011.

[31] B. Hintermann, A. Barg, M. Knupp, and V. Valderrabano, "Conversion of painful ankle arthrodesis to total ankle arthroplasty," *Journal of Bone and Joint Surgery Series A*, vol. 91, no. 4, pp. 850–858, 2009.

[32] M. Horisberger, B. Hintermann, and V. Valderrabano, "Alterations of plantar pressure distribution in posttraumatic end-stage ankle osteoarthritis," *Clinical Biomechanics*, vol. 24, no. 3, pp. 303–307, 2009.

[33] C. L. Saltzman and G. Y. El-Khoury, "The hindfoot alignment view," *Foot and Ankle International*, vol. 16, no. 9, pp. 572–576, 1995.

[34] M. Knupp, G. I. Pagenstert, A. Barg, L. Bolliger, M. E. Easley, and B. Hintermann, "SPECT-CT compared with conventional imaging modalities for the assessment of the varus and valgus malaligned hindfoot," *Journal of Orthopaedic Research*, vol. 27, no. 11, pp. 1461–1466, 2009.

[35] G. I. Pagenstert, A. Barg, A. G. Leumann et al., "SPECT-CT imaging in degenerative joint disease of the foot and ankle," *Journal of Bone and Joint Surgery Series B*, vol. 91, no. 9, pp. 1191–1196, 2009.

[36] S. Brunner, M. Knupp, and B. Hintermann, "Total ankle replacement for the valgus unstable osteoarthritic ankle," *Techniques in Foot and Ankle Surgery*, vol. 9, no. 4, pp. 165–174, 2010.

[37] B. S. Kim, W. J. Choi, Y. S. Kim, and J. W. Lee, "Total ankle replacement in moderate to severe varus deformity of the ankle," *Journal of Bone and Joint Surgery Series B*, vol. 91, no. 9, pp. 1183–1190, 2009.

[38] B. S. Kim and J. W. Lee, "Total ankle replacement for the varus unstable osteoarthritic ankle," *Techniques in Foot and Ankle Surgery*, vol. 9, no. 4, pp. 157–164, 2010.

[39] M. Knupp, S. A. S. Stufkens, L. Bolliger, S. Brunner, and B. Hintermann, "Total ankle replacement and supramalleolar osteotomies for malaligned osteoarthritic ankles," *Techniques in Foot and Ankle Surgery*, vol. 9, no. 4, pp. 175–181, 2010.

[40] M. Knupp, L. Bolliger, A. Barg, and B. Hintermann, "Total ankle replacement for varus deformity," *Orthopade*, vol. 40, no. 11, pp. 964–970, 2011.

[41] M. Knupp, S. A. S. Stufkens, L. Bolliger et al., "Classification and treatment of supramalleolar deformities," *Foot and Ankle International*, vol. 32, pp. 1023–1031, 2011.

[42] N. Gougoulias, A. Khanna, and N. Maffulli, "How successful are current ankle replacements? A systematic review of the literature," *Clinical Orthopaedics and Related Research*, vol. 468, no. 1, pp. 199–208, 2010.

[43] B. T. S. Fevang, S. A. Lie, L. I. Havelin, J. G. Brun, A. Skredderstuen, and O. Furnes, "257 ankle arthroplasties performed in Norway between 1994 and 2005," *Acta Orthopaedica*, vol. 78, no. 5, pp. 575–583, 2007.

[44] A. Henricson, A. Skoog, and A. Carlsson, "The Swedish ankle Arthroplasty Register: an analysis of 531 arthroplasties between 1993 and 2005," *Acta Orthopaedica*, vol. 78, no. 5, pp. 569–574, 2007.

[45] A. H. Hosman, R. B. Mason, T. Hobbs, and A. G. Rothwell, "A New Zealand national joint registry review of 202 total ankle replacements followed for up to 6 years," *Acta Orthopaedica*, vol. 78, no. 5, pp. 584–591, 2007.

[46] E. T. Skyttä, H. Koivu, A. Eskelinen, M. Ikävalko, P. Paavolainen, and V. Remes, "Total ankle replacement: a population-based study of 515 cases from the Finnish Arthroplasty Register," *Acta Orthopaedica*, vol. 81, no. 1, pp. 114–118, 2010.

[47] N. A. Abidi, G. S. Gruen, and S. F. Conti, "Ankle arthrodesis: indications and techniques," *The Journal of the American Academy of Orthopaedic Surgeons*, vol. 8, no. 3, pp. 200–209, 2000.

[48] A. Mohamedean, H. G. Said, M. El-Sharkawi, W. El-Adly, and G. Z. Said, "Technique and short-term results of ankle arthrodesis using anterior plating," *International Orthopaedics*, vol. 34, no. 6, pp. 833–837, 2010.

[49] I. S. Tarkin, M. A. Mormino, M. P. Clare, H. Haider, A. K. Walling, and R. W. Sanders, "Anterior plate supplementation increases ankle arthrodesis construct rigidity," *Foot and Ankle International*, vol. 28, no. 2, pp. 219–223, 2007.

[50] D. E. Bonasia, F. Dettoni, J. E. Femino, P. Phisitkul, M. Germano, and A. Amendola, "Total ankle replacement: why, when and how?" *The Iowa Orthopaedic Journal*, vol. 30, pp. 119–130, 2010.

[51] M. E. Easley, S. B. Adams Jr., W. C. Hembree, and J. K. DeOrio, "Results of total ankle arthroplasty," *Journal of Bone and Joint Surgery Series A*, vol. 93, no. 15, pp. 1455–1468, 2011.

[52] A. J. Guyer and E. G. Richardson, "Current concepts review: total ankle arthroplasty," *Foot and Ankle International*, vol. 29, no. 2, pp. 256–264, 2008.

[53] B. Hintermann and V. Valderrabano, "Total ankle replacement," *Foot and Ankle Clinics*, vol. 8, no. 2, pp. 375–405, 2003.

[54] V. Valderrabano, G. I. Pagenstert, and B. Hintermann, "Total ankle replacement—three-component prosthesis," *Techniques in Foot & Ankle Surgery*, vol. 2, pp. 84–90, 2005.

[55] B. Hintermann, "Total ankle replacement in Switzerland," *Orthopade*, vol. 40, p. 963, 2011.

[56] A. Barg, T. Suter, L. Zwicky, M. Knupp, and B. Hintermann, "Medial pain syndrome in patients with total ankle replacement," *Orthopade*, vol. 40, pp. 991–999, 2011.

[57] K. T. Lee, Y. K. Lee, K. W. Young et al., "Perioperative complications of the MOBILITY total ankle system: comparison with the HINTEGRA total ankle system," *Journal of Orthopaedic Science*, vol. 15, no. 3, pp. 317–322, 2010.

[58] M. S. Myerson and K. Mroczek, "Perioperative complications of total ankle arthroplasty," *Foot and Ankle International*, vol. 24, no. 1, pp. 17–21, 2003.

[59] C. L. Saltzman, A. Amendola, R. Anderson et al., "Surgeon training and complications in total ankle arthroplasty," *Foot and Ankle International*, vol. 24, no. 6, pp. 514–518, 2003.

[60] B. G. Schutte and J. W. K. Louwerens, "Short-term results of our first 49 Scandanavian Total Ankle Replacements (STAR)," *Foot and Ankle International*, vol. 29, no. 2, pp. 124–127, 2008.

[61] H. Kofoed and A. Lundberg-Jensen, "Ankle arthroplasty in patients younger and older than 50 years: a prospective series with long-term follow-up," *Foot and Ankle International*, vol. 20, no. 8, pp. 501–506, 1999.

[62] M. P. Clare and R. W. Sanders, "Preoperative considerations in ankle replacement surgery," *Foot and Ankle Clinics*, vol. 7, no. 4, pp. 709–720, 2002.

[63] H. E. Groth and H. F. Fitch, "Salvage procedures for complications of total ankle arthroplasty," *Clinical Orthopaedics and Related Research*, no. 224, pp. 244–250, 1987.

[64] M. R. McGuire, R. F. Kyle, R. B. Gustilo, and R. F. Premer, "Comparative analysis of ankle arthroplasty vesus ankle

arthrodesis," *Clinical Orthopaedics and Related Research*, no. 226, pp. 174–181, 1988.

[65] M. Nagashima, H. Takahashi, S. Kakumoto, Y. Miyamoto, and S. Yoshino, "Total ankle arthroplasty for deformity of the foot in patients with rheumatoid arthritis using the TNK ankle system: clinical results of 21 cases," *Modern Rheumatology*, vol. 14, no. 1, pp. 48–53, 2004.

[66] F. Shinomiya, M. Okada, Y. Hamada, T. Fujimura, and D. Hamada, "Indications of total ankle arthroplasty for rheumatoid arthritis: evaluation at 5 years or more after the operation," *Modern Rheumatology*, vol. 13, no. 2, pp. 153–159, 2003.

[67] P. L. R. Wood, L. A. Crawford, R. Suneja, and A. Kenyon, "Total ankle replacement for rheumatoid ankle arthritis," *Foot and Ankle Clinics*, vol. 12, no. 3, pp. 497–508, 2007.

[68] A. A. Spirt, M. Assal, and S. T. Hansen, "Complications and failure after total ankle arthroplasty," *Journal of Bone and Joint Surgery Series A*, vol. 86, no. 6, pp. 1172–1178, 2004.

[69] A. Barg, M. Knupp, H. B. Henninger, L. Zwicky, and B. Hintermann, "Total ankle replacement using HINTEGRA—an unconstrained, three-component system: surgical technique and pitfalls," *Foot and Ankle Clinics*. In press.

[70] L. A. DiDomenico, J. R. Treadwell, and L. Z. Cain, "Total ankle arthroplasty in the rheumatoid patient," *Clinics in Podiatric Medicine and Surgery*, vol. 27, no. 2, pp. 295–311, 2010.

[71] V. Gibson and D. Prieskorn, "The valgus ankle," *Foot and Ankle Clinics*, vol. 12, no. 1, pp. 15–27, 2007.

[72] N. E. Gougoulias, A. Khanna, and N. Maffulli, "History and evolution in total ankle arthroplasty," *British Medical Bulletin*, vol. 89, no. 1, pp. 111–151, 2009.

[73] J. M. Murnaghan, D. S. Warnock, and S. A. Henderson, "Total ankle replacement. Early experiences with STAR prosthesis," *Ulster Medical Journal*, vol. 74, no. 1, pp. 9–13, 2005.

[74] J. S. Park and K. J. Mroczek, "Total ankle arthroplasty," *Bulletin of the NYU Hospital for Joint Diseases*, vol. 69, no. 1, pp. 27–35, 2011.

[75] C. L. Saltzman, "Perspective on total ankle replacement," *Foot and Ankle Clinics*, vol. 5, no. 4, pp. 761–775, 2000.

[76] P. L. R. Wood, T. M. Clough, and R. Smith, "The present state of ankle arthroplasty," *Foot and Ankle Surgery*, vol. 14, no. 3, pp. 115–119, 2008.

[77] B. Hintermann, *Endoprothetik des Sprunggelenks: Historischer Überblick, Aktuelle Therapiekonzepte und Entwicklungen*, Springer, Wien, Austria, 2005.

[78] V. Valderrabano, B. Hintermann, V. Von Tscharner, B. Göpfert, W. Dick, and B. Nigg, "Muscle biomechanics in total ankle replacement," *Orthopade*, vol. 35, no. 5, pp. 513–520, 2006.

[79] V. Valderrabano, B. M. Nigg, V. Von Tscharner, C. B. Frank, and B. Hintermann, "Total ankle replacement in ankle osteoarthritis: an analysis of muscle rehabilitation," *Foot and Ankle International*, vol. 28, no. 2, pp. 281–291, 2007.

[80] V. Valderrabano, V. von Tscharner, B. M. Nigg, B. Goepfert, C. B. Frank, and B. Hintermann, "Lower leg muscle atrophy in ankle osteoarthritis and its rehabilitation after implantation of a total ankle arthroplasty," *Fuss und Sprunggelenk*, vol. 5, no. 1, pp. 33–43, 2007.

[81] A. Barg and B. Hintermann, "Implantate und biomechanik des oberen sprunggelenks," in *AE-Manual der Endoprothetik: Sprunggelenk und Fuss*, W. Neumann, Ed., pp. 29–45, Springer, London, UK, 2012.